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A Synopsis of Known and Potential Diseases and Parasites Associated with Climate Change

Ontario

Ontario Forest Research Institute

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A Synopsis of Known and Potential Diseases and Parasites Associated With Climate Change

Compiled by

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2003

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Abstract

Current climate change models for Ontario predict that mean annual temperature will increase by up to 3°C over the next century. Temperatures are expected to increase more in winter than in summer, with an increase in frost-free days, and to increase more in northern than southern latitudes. Minimum temperatures should increase more than maximum temperatures. Precipitation, although somewhat unpredictable, is expected to increase in Ontario, but increased evaporation and transpiration due to warmer temperatures will likely result in drier conditions, particularly during the summer growing seasons. If Ontario's climate changes as predicted, many human, animal and plant diseases that are affected by climate will also change in range, intensity, and/or a variety of other ways, in turn affecting the health and well-being of people, animals and plants in Ontario. This report identifies which diseases are likely to be affected by climate and which human, animal, and plant groups are most vulnerable to them. It also provides recommendations for changing disease management strategies and practices to mitigate the effects of climate change. Because we cannot predict precisely how climate change will affect disease and because the predicted changes may occur at a relatively fast pace, we must improve our knowledge base and our response capability at local, regional and national levels to maintain current levels of health. Doing so will enable us to face the coming challenges, adapt to them, learn from them, and protect Ontario's communities and ecosystems.

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Structure of This Report

The main body of the report is prefaced by a popular summary that provides a brief overview of the effects of climate change on disease in Ontario.

The main body of this report is divided into 2 sections, titled *A Synopsis of Known and Potential Diseases and Parasites of Humans and Animals Associated With Climate Change in Ontario* and *Climate Change and Plant Disease in Ontario*. Each section has its own table of contents and page numbering system.

Popular summary

Introduction

Diseases, be they of humans, animals, or plants, play important roles in how all life on Earth evolves and develops. They have influenced the social and economic structure and indeed the very existence of past and present civilizations. Many diseases, and the organisms that cause them, are integral components of intricate natural ecosystems, involved in such varied processes as nutrient cycling in forests and the succession of different plant species on a landscape. The results of climate change (for example, an increase in temperature) may affect diseases in many ways; however, the complexity of the links between diseases and the systems in which they exist makes it difficult to predict these changes. Climate (only one of the many determinants of health) interacts in complex and dynamic ways with the biophysical and social environment in shaping individual and population health.

Climate Change

Ontario's climate is undergoing significant change, as climate patterns worldwide react to the accumulation of greenhouse gases in the atmosphere. Climate change models predict the extent of these changes into the future. For Ontario, overall predictions include:

- *Warmer temperatures.* Over the next 100 years, the average annual temperature is expected to increase by 3°C. This increase will affect average winter temperature more than summer temperature. Although these changes seem slight, their effects are greatly magnified at extreme values. In general, weather will become more variable under climate change.
- *Changes in rain- and snowfall.* Over much of Ontario, precipitation is expected to increase, although regional changes are difficult to predict. Increased evaporation and transpiration (i.e., water loss from living things) because of warmer temperatures may actually lead to drier conditions, especially during the summer.
- *Increased frequency of extreme weather events.* Drought, rain, hail, and ice and wind storms are predicted to increase in frequency. For example, an event that now has a probability of occurring every 30 years may begin to occur every 4 or 5 years.

Because climate affects so many different elements and processes in the ecology of diseases, their response to specific climate change scenarios is difficult to predict. However, we can start by examining what we already understand about the relationships between climate and disease. In this way we can begin identifying those diseases that climate change is most likely to affect. By combining what we understand about climate and disease with output from climate change model projections, a range of possible future disease issues become apparent. Although such projected impacts of future climate on disease patterns are primarily informed conjecture, they are the best that is available to us now and are essential to planning how to adapt to climate change.

Disease – What Is It?

A *disease* is a condition that impairs the proper function of any living thing or *organism.* A disease can affect an entire organism or just one part. Diseases can be caused by many different things, including other living organisms (bacteria, fungi, and viruses) and non-living causes (excessive heat, cold, drought). Some diseases are caused by a combination of both living and non-living factors.

The relationships between the disease-causing *agents*, the organism affected by the disease (termed a *host*), and the *environments* in which they function are often depicted as a "disease triangle" (Figure 1). For each disease, all 3 points of the triangle must exist together in space and time for disease to occur. Often, multiple hosts, agents, and environments interact. For example, Lyme disease requires multiple hosts, vectors, and diverse environments; its spread into northern Ontario is limited by the environmental requirements of the virus-carrying ticks. Without the right environment "point" on the triangle, Lyme disease cannot occur. Some of the most severe and unpredictable consequences of climate change with respect to disease would occur if populations of disease agents and hosts, which were formerly geographically separated due to climate constraints, converged.

Disease can also be measured at the group or population level: In a herd of cattle with tuberculosis, not all cows may have the disease. Disease can also be measured at community and ecosystem levels: A healthy rural community, a diseased forest, a dead lake. Adapting

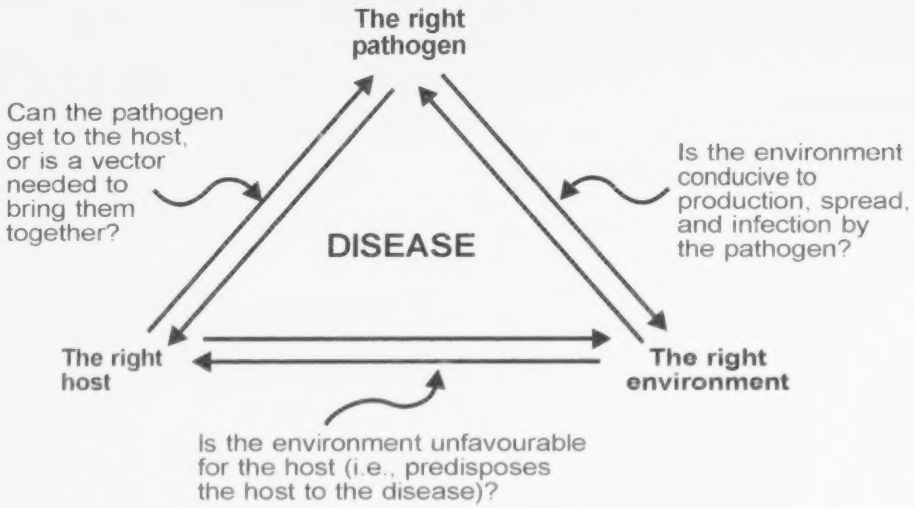
The right pathogen
Can the pathogen get to the host, or is a vector needed to bring them together?
Is the environment conducive to production, spread, and infection by the pathogen?
DISEASE
The right host
The right environment
Is the environment unfavourable for the host (i.e., predisposes the host to the disease)?

Figure 1. *The disease triangle is a simplified view of how diseases work.*

to health impacts of climate change can occur at many different scales, such as individuals protecting themselves against mosquito bites or applying larvicides to standing water where mosquitoes breed. An adaptation plan is formulated with a range of possible adaptive responses.

Many factors in combination determine whether or not individual organisms or communities are affected by disease. Climate change may affect one or more of these factors in a variety of ways, making it difficult to predict actual outcomes. For example, predicted milder winters may increase the survival of the fungus responsible for white mould, a very important disease of beans, canola, carrots, lettuce, and soybeans. However, warmer, drier conditions over the growing season will reduce infection by this disease. Therefore, climate change may affect a disease system both positively and negatively.

Climate Change and Disease

Climate change factors can *directly* cause disease. For example, if Ontario's climate changes as predicted, humans, animals, and plants will likely experience more heat stress due to more frequent extreme heat waves. These direct links between climate and disease are relatively easy to identify. Probably more important, however, are the complex *indirect* effects of climate change on diseases (Figure 2). These include effects on the organisms that cause disease, on the organisms that carry disease to the host (called *vectors*), on the animals or plants that "store" disease (called *disease reservoirs*), and on the host itself. These multiple effects can be difficult to identify and predict.

Direct Effects of Climate Change

Diseases that can be directly affected by climate change are those that are associated with chemical and physical factors such as temperature or moisture extremes or air pollution. For example:

- More heat waves may lead to increased illness and death of people, animals, and plants. For humans, the greatest impact of heat waves occurs in large cities because of the "heat island" effect. In the Toronto-Niagara region, annual heat-related deaths of the elderly could reach 447 by 2020 in an average summer.
- Milder winter temperatures could reduce the risk of hypothermia and hypoglycemia in newborn animals such as lambs.
- Increased carbon dioxide (CO_2) levels may stimulate growth of some fungi that cause plant diseases.
- Increased ozone and other pollutants will reduce plant vitality and affect respiratory diseases of humans and animals.

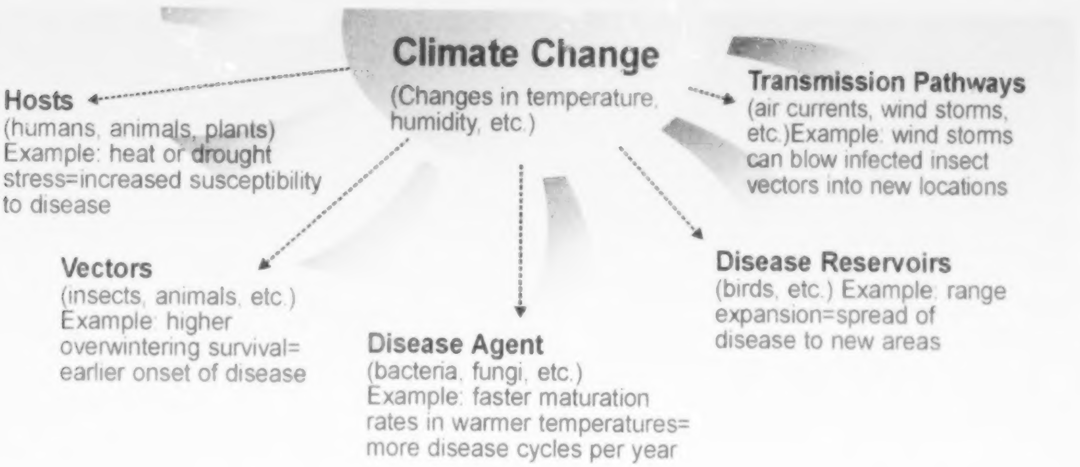
Climate Change
(Changes in temperature, humidity, etc.)
Hosts
(humans, animals, plants)
Example: heat or drought stress=increased susceptibility to disease
Transmission Pathways
(air currents, wind storms, etc.)Example: wind storms can blow infected insect vectors into new locations
Vectors
(insects, animals, etc.)
Example: higher overwintering survival= earlier onset of disease
Disease Reservoirs
(birds, etc.) Example: range expansion=spread of disease to new areas
Disease Agent
(bacteria, fungi, etc.)
Example: faster maturation rates in warmer temperatures= more disease cycles per year

Figure 2. *The effects of climate change on the many components that make up a disease system can be positive or negative.*

Indirect Effects of Climate Change

Organisms that cause disease include bacteria, fungi, viruses, nematodes, phytoplasmas, and parasitic plants. These disease agents are called *pathogens*. Bacteria and viruses are the main causes of infectious diseases in humans and other animals, whereas fungi cause the majority of plant diseases.

A. Infectious Diseases of Humans and Animals

Weather and climate have long been known to affect the health of humans and animals. Often, diseases of humans and animals are intimately connected. Many of the bacteria, viruses, and parasites that infect animals can be transmitted to people (e.g., rabies, West Nile virus, *E. coli*). These diseases are called *zoonoses*. They can be transferred from one animal species to another, including humans. Zoonotic disease outbreaks in animals can serve as early warnings for human disease problems.

Diseases can be transmitted to the host animal in a number of ways (Table 1). Insects often carry diseases to host animals (i.e., they are vectors). Because insects are cold-blooded, their feeding activity, reproductive success, survival, and ability to transmit disease are directly determined by temperature and humidity levels. Vectorborne diseases that are not common in Ontario today may become more prevalent in a warmer climate. For example, mosquitoes infected with viruses that cause eastern equine or St. Louis encephalitis could be carried on birds or by wind into Canada from the United States, and, under appropriate climatic conditions, cause disease outbreaks.

Most food- and waterborne diseases are zoonotic, with reservoirs in livestock and wildlife, from which they spread to water and foods. Food- and waterborne illnesses pose a significant health burden: In Canada, the annual cost of foodborne disease in humans has been estimated at $1.3 billion in 1985 dollars. The incidence of foodborne disease peaks in summer, as hot weather promotes pathogen growth in food, and people's behaviour (picnics, barbecues) favours disease transmission. Warm, humid conditions favour growth of fungi on crops used for animal and human consumption. Livestock become ill when fungi infecting their feed produce dangerous mycotoxins (poisons). Heavy rainfall can also lead to disease outbreaks. Over half of the waterborne disease outbreaks in the United States in the latter half of the 20th century were preceded by an extreme precipitation event. In Ontario, the E. coli O157:H7 water contamination at Walkerton in 2000 was preceded by a severe rain storm. When drought is followed by heavy rain, contamination is flushed through watersheds, resulting in greater probability of disease outbreaks. This type of contamination may dramatically affect fish, especially farmed fish.

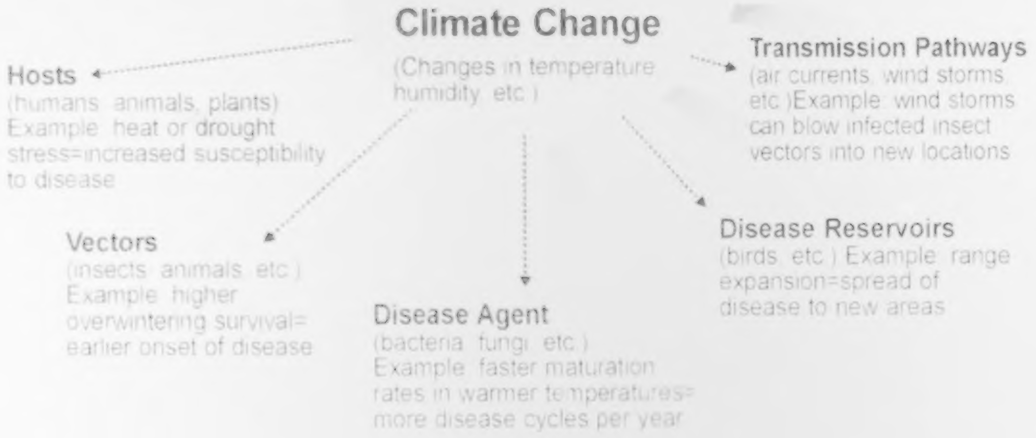
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Figure 2. The effect of climate change on the disease [illegible]

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Table 1. *Modes of disease transmission to humans and animals.*

Transmission Mode	Examples	Possible Effects of Climate Change
Vector	West Nile virus Lyme disease	Lengthened transmission season, increased overwinter survival, range expansion, more frequent transmission
Food	Salmonella Mycotoxicosis in pigs	Increased risk of food contamination, increased replication and survival of pathogens, higher incidence of pathogens in animal reservoirs
Water	E. coli O157:H7 Campylobacter	Increased risk of outbreaks because of extreme precipitation events
Direct	Rabies	Alterations in ranges and population dynamics of animal reservoirs (e.g., fox, raccoon)

The mild winters predicted with climate change could also result in increased parasite loads in horses, livestock, dogs, and cats.

B. Plant Diseases Caused by Biotic Agents

At first glance, one might expect plants to benefit from the warmer temperatures and elevated levels of atmospheric CO_2 predicted with climate change. However, plants will also be stressed by drier soils and more extreme weather, resulting in increased disease susceptibility.

For plants, there will probably be more changes in the types of diseases that develop than their average annual severity. Historically, Ontario's long, cold winters have reduced survival of organisms that cause disease, restricted the number of disease generations per year, and limited disease activity during the growing season. If Ontario's climate warms, these issues of disease survival, growth, and activity will change. For disease survival, climate change effects will depend on the way in which the pathogen presently survives adverse conditions (Table 2).

Climate change will also affect how plant diseases develop during the growing season. Warmer, drier summers are expected to decrease diseases where moisture and then temperature are the most important factors in the development of plant disease epidemics. However, increased hail, rain, and wind storms will increase the risk of disease infection through plant wounds. Interactions between diseases caused by pathogens and non-living agents are also anticipated and may represent the most important effects of climate change on plant diseases in Ontario and elsewhere.

Table 2. *Possible effects of climate change on plant pathogen survival.*

Survival Mode of Pathogen	Common Types of Pathogens	Effects of Climate Change
Disease organisms that survive in soil	Fungi and bacteria	Thick-walled spores and other survival structures produced by these pathogens should not be greatly affected by climate change
Disease organisms that survive in plants or plant debris	Fungi and bacteria	Milder winters could increase survival of pathogens that overwinter on living and dead plants
Disease organisms that are transferred by vectors	Viruses and fungi	Milder winters could increase survival of insect vectors; milder summers could result in increased development and reproductive rates
Introduced diseases	Fungi and bacteria	Pathogens that do not presently survive Ontario winters may do so if winters become milder

Mitigation and Adaptation Strategies

The climate of Ontario is changing, resulting in changes in the human, animal, and plant diseases that we need to prevent or manage. Because climate change predictions are based on uncertain information, especially at regional and local levels, our response must be in the form of adaptive strategies that are reviewed and adjusted as new information and improved climate models become available. Developing effective strategies will require the involvement of government agencies, academia, and the general public.

1. *Enhanced Surveillance.* Diagnostic tools and personnel are needed to detect new diseases in Ontario. These newly introduced diseases can have devastating effects on human, animal, or plant communities. One such monitoring system is the West Nile virus surveillance program, which is based on monitoring animal sentinels (mostly crows) and vectors (mosquitoes). This system is an example of a coordinated federal and provincial surveillance program involving public health officials and wildlife and animal health professionals.

Other national disease surveillance agencies include:

- the Canadian Food Inspection Agency (livestock and plant diseases)
- the Canadian Animal Health Network (livestock diseases)
- Flu Watch (human disease)
- the National Enteric Surveillance Program (human disease)
- the Canadian Enteric Outbreak Centre (human disease)

Increased surveillance by these and other agencies will be necessary if climate change results in more disease introductions into Ontario.

2. *Enhanced Research and Development.* To date, little attention has been given to the effects of climate change on plant or animal health. For plant diseases, the rapidity of the predicted climate change will necessitate more research into alternative crops and cultivars with increased stress tolerance and disease resistance, enhanced cultural practices, and climate-based site selection. Farmers have learned to protect their animals from weather and seasonal changes; however, enhanced structural and cooling/heating systems may be necessary to protect animals against extreme weather events. Developing information and research networks will play a vital role by linking researchers and practitioners across the country to allow information to be gathered, reviewed, and redistributed to stakeholders. Seven national networks on human health and climate change have already been created by the Health Canada Climate Change and Health Office. They range in theme from temperature-related illness to social and economic impacts of climate change on health.

3. *Enhanced Public and Health Professional Awareness.* Public awareness is increasing about the health hazards of mosquito bites related to West Nile virus. Similar approaches can be taken regarding tickborne diseases, water contamination, and foodborne illnesses. The Health Canada Climate Change and Health web site is a public source of climate-related health information. Continuing education on climate-related disease issues would help human and animal health care professionals and plant disease specialists gain the skills to detect and identify newly emerging problems.

4. *Integrated and Adaptive Policy Development.* Mitigating and adapting to climate change will only be successful in a supportive and encouraging policy environment in many varied fields, including agriculture, natural resource management, and human health.

Conclusion

We can identify some of the diseases that are most likely to be affected by climate change, and which human, animal, or plant groups may be most vulnerable, but we are unlikely to be able to predict the exact nature of the hazard. Improving our knowledge base and our response capability at local, regional and national levels will enable us to face the coming challenges, adapt to them, learn from them, and continue to maintain healthy communities and ecosystems in Ontario.

Case Study 1 – Influenza Type A

Causal agent: virus
Vector: None
Intermediate hosts: pigs, aquatic animals
Hosts: birds, humans, pigs, and other animals

Influenza A is an example of a zoonotic disease – one that is transferred directly from animals to other animals, including humans. The virus strains that cause influenza are usually very specific to one certain species of animal. For example, the influenza virus in birds is not the same virus that causes influenza in humans. However, other animals, especially pigs, play important roles in the development of human influenza viruses. The genes for all influenza viruses are contained in the influenza carried by wild aquatic birds – these birds do not get sick from the virus and are the reservoir of the virus in nature. Pigs are susceptible to influenza viruses of both birds and mammals. These intermediate hosts act as mixing vessels, where bird and mammalian viruses are mixed to generate new viral strains, which can be transferred to other animals. Aquatic animals such as seals also appear to be susceptible to avian influenza viruses and may be an important link in the evolution of new mammalian strains.

Although the emergence of new influenza viral strains is usually linked to the climate and agricultural systems of Asia, influenza outbreaks in poultry and swineherds in North America have recently occurred. Climate changes that alter behaviour and infection rates in wild bird populations (e.g., overwintering patterns and migration routes), combined with changes in animal management (e.g., outdoor rearing and increased ventilation during heat waves) may increase the likelihood of viral transfer and infection in domestic animals in Ontario. Similarly, changes in ocean coastal temperature could influence influenza occurrence in seals and other marine animals.

Case Study 2 - West Nile Virus

Causal Agent: virus
Vector: mosquito (*Culex pipiens*)
Reservoir: wild birds
Hosts: wild birds including blue jays and crows, humans, horses

West Nile virus (WNV) is found in many European, Middle Eastern, Asian, and African countries. In 1999, it was detected for the first time in North America, and in 2001, the virus was identified in dead birds in southern Ontario. The first human cases in Ontario were reported in late summer 2002. Mosquitoes usually transmit WNV among songbirds, which often do not show any signs of disease. Birds in the Corvidae family (e.g., American crows, blue jays) and raptors appear to be highly susceptible to the disease. A cycle of transmission between wild birds and native mosquitoes has evidently become established in North America, with large numbers of crows dying. Mosquitoes that pick up the virus from infected birds and mammals can transmit the disease to humans and horses.

Projected climate change in Ontario may contribute to the establishment and spread of WNV. Climatic conditions, including a mild winter coupled with prolonged drought and heat waves, may have played a role in the initial outbreak in North America and the early onset of transmission in Ontario in 2002. Mosquitoes are better vectors for WNV during periods of high temperatures, and the virus appears to be able to overwinter in Ontario during mild winters. WNV is an example of an exotic disease that has recently become established in Ontario, perhaps partly due to a changing climate.

Case Study 3 – Stewart's Wilt of Corn

Causal agent: bacteria
Vector: corn flea beetle
Hosts: corn, especially sweet corn

This bacterial disease is transferred to corn plants throughout the growing season by the corn flea beetle. The disease can reduce corn yields from 40-100% on infected plants.

The bacteria overwinters in the gut of the flea beetle; therefore, milder winters would increase survival of both beetles and the bacteria. The mild winters in 1998 and 1999 led to the most severe outbreak of Stewart's wilt recorded in Ontario. Dry summer weather favours beetle multiplication and feeding. Disease severity generally increases in high temperatures. If Ontario's climate changes as predicted, Stewart's wilt is likely to increase.

Case Study 4 - Maple Decline

Host: maple trees
Predisposing factors: site characteristics, climate
Inciting factors: years of drought or heavy rain
Contributing factors: diseases and insect feeding

Maple decline is a "disease complex" that results in a general deterioration in tree health over a large area. Affected trees grow slower, their branches die, and many trees die outright. No single event or stress leads to decline, rather a combination of several interacting factors such as site characteristics, climate, weather extremes, diseases, and insect feeding progressively stress individual trees until they weaken and die. Stressed trees become susceptible to pests such as wood boring beetles, decay, and root rots.

Under climate change, increased temperature and evapotranspiration, as well as increased frequency of extreme weather events, will increase the frequency and severity of stress factors leading to maple and other forest declines.

A Synopsis of Known and Potential Diseases and Parasites of Humans and Animals Associated With Climate Change in Ontario

A Synopsis of Known and Potential Diseases and Parasites of Humans and Animals Associated With Climate Change in Ontario

by

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Dominique Charron

David Waltner-Toews

Preface

Weather and climate have long been known to affect the health of people and animals either directly (e.g., as a result of temperature extremes or other hazardous weather and climatic events) or indirectly through complex social and ecological mechanisms impacting a wide variety of infectious agents present in nature. Global climate change is expected to alter the dynamics of these health impacts to varying degrees. These reports were commissioned by the Ontario Ministry of Natural Resources to provide a synoptic overview of known and potential diseases and parasites in Ontario, the species of animals that they affect, the possible impacts of climate change, and known control and management tools and techniques.

Diseases are classified in a variety of ways. In terms of climate change impacts, we can think of diseases that affect primarily people and those that affect primarily other animals. This report is divided into two sections to reflect this difference in emphasis. Dr. Charron (Health Canada) is lead author on the human report, and Dr. Waltner-Toews (University of Guelph) is the lead on the animal report. These reports focus on potential climate change impacts most relevant to the province of Ontario. It was not within the scope of this report to analyze data or to perform formal risk assessments of the impact of climate change on disease. Rather, the authors have offered an inventory of the human and animal diseases in Ontario that could be affected by climate change and discussed *how* they might be affected. The authors have also provided a discussion on adaptation strategies that will help Ontarians prepare for the potential health impacts of climate change, despite a great deal of uncertainty around precisely what those impacts will be.

Climate change will affect people and animals by acting through the eco-social systems of which they are a part. Many health outcomes are the result of various relationships with infectious agents (viruses, bacteria, parasites, and fungi). Many of these agents are *zoonoses*, i.e., they are transmitted to people by animals. Some are transmitted through contamination of food, water, or the general environment; some move via vectors such as mosquitoes; and only a few are transmitted by more direct routes, such as animal bites and scratches. Many of these infectious agents cause diseases in animals as well. Therefore, zoonoses appear in both the animal section and the human section of this report, with a somewhat different emphasis in each case. The preface, executive summaries and outline, as well as the sections on adaptation and mitigation measures, the list of consultations, and the appendices, are common to both the animal and human sections of the report. A full understanding of the effects of climate change on these diseases cannot be achieved without reference to all parts of the report.

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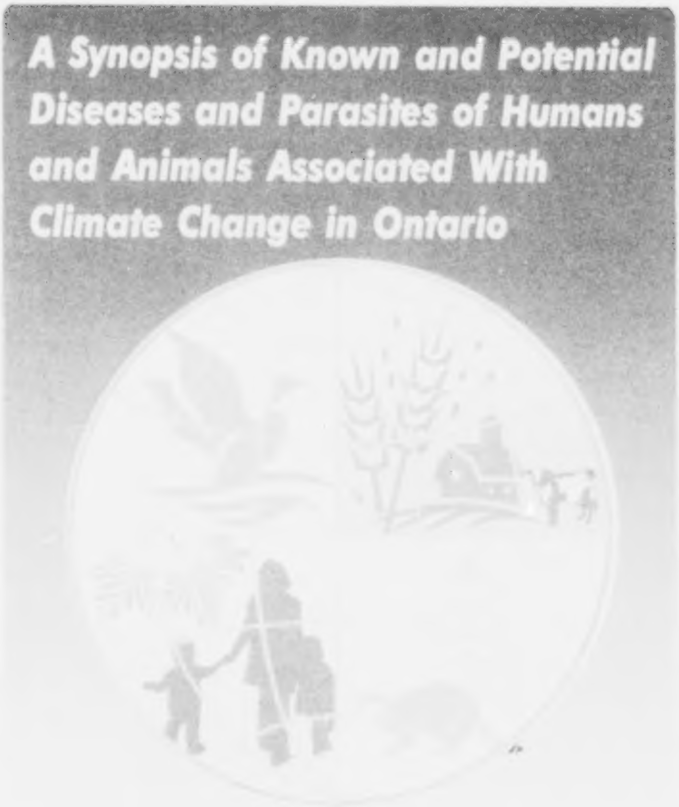

A Synopsis of Known and Potential Diseases and Parasites of Humans and Animals Associated With Climate Change in Ontario

Part A.

Executive Summaries

Executive Summary: Diseases in People

Infectious disease threats to Ontarians are not new: Influenza, sexually transmitted diseases, foodborne and waterborne infections and the common cold are modern daily occurrences. Historically, tuberculosis, typhoid, and influenza exacted a heavy toll in Ontario. Then as now, Ontario's public health system invests considerable effort in preventing and controlling these and other infectious diseases. Because of public health care systems, proper treatment is now easily available for those affected. Timely treatment also helps control disease spread. Because of these systems, and related ones such as sewage treatment, food safety, and drinking water treatment, Ontarians enjoy excellent protection from infectious diseases relative to most places in the world. All systems, including water treatment systems and public health systems, operate within certain norms and constraints, including weather, or more generally, climate. There is now considerable evidence that Ontario's normal climate will undergo significant change, as climate patterns worldwide react to the accumulation of greenhouse gases in the atmosphere. Despite our best efforts in curbing greenhouse gas emissions, the global average temperature is likely to warm by between 2 and 5 degrees Celsius by the year 2100 (IPCC 2001).

Climate affects the health of people in Ontario every day. Asthma sufferers, the elderly and others with respiratory problems are affected by heat waves and smog. All Ontarians worry about frostbite and exposure in the winter. Influenza occurs seasonally in fall and winter in Ontario. Some Ontario communities are acutely aware of the potential threat to drinking water posed by heavy rainfall. Weather and climate also affect the geographic distribution and level of risk of many other infectious diseases.

Many infectious diseases have animal reservoirs or insect vectors. The pathogens, animals and insects are directly affected by temperature, humidity, and other environmental factors. Examples include rabies, tick-borne Lyme disease, the waterborne protozoa *Cryptosporidium parvum* and *Giardia lamblia*, and deadly *Escherichia coli* O157 made infamous by the Walkerton tragedy. It is generally assumed that Ontario's temperate climate (warm summer/cold winter) is not generally conducive to many of the mosquito-borne diseases (malaria, dengue fever) that are much more common in the tropics. However, the rapid spread of mosquito-borne West Nile virus suggests that this assumption may need to change.

Climate change predictions for Ontario include warmer temperatures, more frequent extreme weather events, poorer air quality, and changes in precipitation patterns. These changes may alter the risk of many illnesses, including heat stress, respiratory disease, vectorborne diseases, foodborne and waterborne illnesses, and stress-related problems due to social and economic disruption. However, many factors in combination determine whether or not individuals or communities are affected by disease. Other health determinants include individual susceptibility (immunity, age, occupation), social and economic constraints, and geography. From agriculture to urbanization, various human modifications of the environment interact with ecological systems. How changes in climate and the weather affect these complex dynamics may result in altered threats to the health of Ontarians.

Table A.1 is a relatively comprehensive list, based on scientific principles and common sense, of the general impacts of climate change on the health of Ontarians. These are classified into direct and indirect effects, according to whether they occur predominantly through the impacts of climate variables on human biology or are mediated by climate-induced changes in other ecological and biogeochemical systems. More detailed information on the potential indirect impacts of climate change on infectious diseases, some extremely rare or non-existent in Ontario, are listed in subsequent tables on vectorborne diseases (Table A.2), zoonoses transmitted by direct contact between humans and animals (Table A.3), foodborne diseases (Table A.4) and waterborne diseases (Table A.5).

In general, diseases that pose a significant health threat to Ontarians today and that appear to be influenced by climate should become top priorities to those preparing a response plan with climate change in mind. These diseases would include heat-related morbidity and mortality, and waterborne and foodborne infections, including *Cryptosporidium* and *E.*

coli O157. Priority should also be given to diseases that may not affect a large number of Ontarians today but that are widespread nearby and could become more active in Ontario because of climate change. Examples include the vectorborne diseases Lyme disease and West Nile virus. The potential for the introduction of exotic diseases (cholera, malaria) should not be neglected.

Ontario needs to prepare a response to the potential health impacts of climate change. This response must consist of adaptive strategies that are reviewed and improved as new information becomes available. In practice, adaptive strategies include public and health professional awareness, enhanced surveillance, interventions within ecosystems, medical and veterinary interventions, infrastructure development, additional research and technology development, and finally, integrated and adaptive policy development. The goal is to achieve public health policy coordinated across all levels of government and across all relevant sectors. Of course, the issues relevant to climate change and public health policy are characterized by conflicting points of view, different stakeholder values, high levels of uncertainty, and a sense of urgency that "something must be done." There is no one set of experts, nor any single method of investigation or paradigm that can capture the truth, nor any single right answer. Therein lies the challenge as we move forward: To identify health threats related to climate change in Ontario, to develop strategies to prevent adverse health outcomes, and to mitigate health impacts where prevention is not possible.

Table A.1. *(Summary table): Potential effects of climate change on human health.*

Climate Parameter	Potential Direct Effects	Potential Indirect Effects
Increased temperatures (particularly nighttime lows, during winter and at northern latitudes)	• Increased morbidity and mortality associated with summer heat stress including heat exhaustion, heatstroke, and deaths associated with respiratory and cardiovascular illness, increased occupational health risks • Reduced morbidity and mortality associated with milder winters	• Alterations in incidence and distribution of vector- and rodent-borne diseases and in direct zoonoses • Range shifts and alterations of population demographics of host, vector and reservoir species due to climate-driven habitat change • Lengthened transmission season for vectorborne disease • Altered pathogen dynamics within host or vector species (for example, accelerated viral extrinsic incubation period) • Alterations in quality/quantity of food supply • Altered crop productivity, nutrient value and potential impacts of plant pathogens and pests • Increased risk for foodborne disease (temperature-dependent bacterial contamination of human food) • Nutritional health risks for Aboriginal communities dependent on local abundance and distribution of wildlife, fish and vegetation
Altered amount and pattern of precipitation		• Increased risk for waterborne disease • Alterations in water quality due to increased agricultural and urban runoff and microbial contamination • Alterations in quality/quantity of food supply
Altered patterns of extreme weather events (convective storms, floods, ice storms, tornadoes)	• Deaths, injuries, stress-related disorders, social disruption, environmentally forced migration • Thunderstorm-related asthma	• Increased risk for waterborne disease with flooding (sewage, animal waste, toxic wastes from disposal sites, agrochemical products) • Risk of communicable diseases due to crowded conditions and poor sanitation in temporary shelters for people displaced by extreme weather • Altered potential for airborne pathogen distribution • Altered airborne vector dispersal pattern
Reduced air quality (including toxicants, acid precipitation, ground-level ozone, particulates)	• Increased morbidity and mortality associated with respiratory disorders, including asthma, allergic rhinitis, COPD and cardiovascular disease	• Immune system suppression leading to increased susceptibility to other diseases • Possible role of some airborne toxicants in cancer
Stratospheric ozone depletion and increased ultraviolet radiation	• Increased incidence of skin cancers, cataracts, effects on immune system	• UV-mediated crop damage and loss, nutritional changes to food • UV-associated photochemical formation of ground-level ozone, contributing to smog

Table A.2. *Potential effects of climate change on vectorborne diseases of humans.*

Disease (Agent)	Reservoir (R), Sentinel (S) and Vector (V)[1]	Current Disease Status[2]	Effects of Climate Variability and Change	Susceptible Populations	References[3]
Bacterial Diseases					
Lyme disease (*Borrelia burgdorferi*)	R: White-footed mouse (*Peromyscus leucopus*), white-tailed deer (*Odocoileus virginianus*), birds, other mice and deer S: Dogs (*Canis domesticus*) V: Black-legged tick (*Ixodes scapularis*) Infected ticks endemic in Long Point, ON; north shore of lake Erie; also occurs in BC; sporadic occurrence SK, MB, ON, NS, QC and NF; disease is rare	Reportable in ON	Northward expansion of range; milder winters increased overwinter survival of vector eggs; warmer weather may increase human exposure due to recreational activities in tick habitat; increased tick habitat if vegetation becomes more lush or dense; increased vertebrate host populations will increase number of infected ticks	People who spend time in grassy and wooded environments, outdoor workers, outdoor sports enthusiasts, dog owners, hunters, hikers	Dworkin et al 1998; Anderson et al. 1994; Artsob et al. 2000; Banerjee et al. 1996, 2000; Barker and Lindsay 2000; Gallivan et al. 1998; Lindsay et al. 1998, 1999; Morshed et al 2000; Varde et al 1998; Lane et al. 1991; CDC 2001b; Steere, 2000; Health Canada 2000; de Mik et al 1997; Klich et al 1996; Wilson 2002; Smith et al 2001; Schmidt et al. 2001

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[1] *Reservoir* refers to the animal or insect population in which infection is maintained. *Vector* refers to the arthropod that transmits the infection between animals and from animals to people. *Sentinel* populations are animal populations that are highly susceptible to infection and can serve as sentinels for human exposure hazard.

[2] *Federally reportable* diseases are nationally notifiable (all cases must be reported to Health Canada). Some provinces conduct surveillance of additional diseases. Diseases that are notifiable in Ontario, but not necessarily in other provinces, are listed as *reportable in ON*.

[3] Full references are listed in Part B of this report.

Table A.2. Cont.

Disease (Agent)	Reservoir (R), Sentinel (S) and Vector (V)[1]	Current Disease Status[2]	Effects of Climate Variability and Change	Susceptible Populations	References[3]
1) **Human monocytic ehrlichiosis** (*Ehrlichia chaffeensis*) 2) **Human granulocytic ehrlichiosis** (*Anaplasma phagocytophila*)	1) R: Dogs, deer, goats (*Capra hircus* V: Lone star tick (*Amblyomma americanum*) 2) R: Deer, wild rodents, elk (*Cervus elaphus*), possibly horses (*Equus equus*), and donkeys (*equus asinus*) V: black-legged tick, western black-legged tick (*I pacificus*)	1) Infected ticks and cases of disease sporadic in southeastern to midwestern U.S. Reportable: no 2) Focally Sporadic in southern ON; eastern seaboard to midwestern U.S., also west of Lake Michigan and West Coast, with a few pockets in the Rockies Reportable: no	Both diseases: northward expansion of range; milder winters increase overwinter survival of vector eggs; warmer weather may increase human exposure due to recreational activities in tick habitat. Increased tick habitat if vegetation becomes more lush or dense; increased vertebrate host populations will increase number of infected ticks; by changes to vegetation, both host and vector habitats will be affected by precipitation - potential for epizootics when naive host population first infected	People who spend time in grassy and wooded environments, outdoor sports enthusiasts, dog owners	Lederberg et al. 1992; CDC website 2001; Varde et al. 1998; Telford et al. 1996; Drebot et al. 2001
Plague (*Yersinia pestis*)	See Table A.3 (rodentborne diseases)				
Rocky Mountain spotted fever (*Rickettsia rickettsii*)	R: Ticks are also the reservoir; dogs and medium-sized mammals are the preferred host for the American dog tick (*Dermacentor variabilis*), and large mammals are preferred by the Rocky Mountain wood tick (*D. Andersoni*) V: American dog tick, Rocky Mountain wood tick, black-legged tick	Focally sporadic in southwestern Canada, southern ON and QC; Continental U.S. Reportable: no	Northward expansion of range; milder winters increase overwinter survival of vector eggs and have other impacts on tick ecology, affecting transmission; warmer weather may increase human exposure due to recreational activities in tick habitat	People who spend time in grassy and wooded environments, outdoor sports enthusiasts, dog owners	Lederberg et al. 1992; CDC website 2001; Drebot et al. 2001; Varde et al. 1998; Walker & Raoult 2000; Lang et al. 1991; Lang 1989; Maurin & Raoult, 1999; Hawker et al. 1998; Wellock, 1960;

Table A.2. Cont.

Disease (Agent)	Reservoir (R), Sentinel (S) and Vector (V)[1]	Current Disease Status[2]	Effects of Climate Variability and Change	Susceptible Populations	References[3]
Tularemia Type A *(Franciscella tularensis)*	R: hares, jack rabbits (*Lepus* spp.) V: tick (*Dermacentor* spp.) or deer fly (*Chrysops* spp., type A only)	Type A disease is extremely rare, though may occur in southern Canada; occurs in southwest U.S. Reportable: in ON	Most cases of tularemia in Canada are Type B and have occurred from direct transmission from infected rabbits; however, tick and deer fly vector ecology will be affected by increased temperature, resulting in potentially greater levels of infection in vectors, prolonged transmission season, and reduced winter die-off; by changes to vegetation,both host and vector habitats will be affected by precipitation; potential for epizootics when naive host population first infected	People who spend time in grassy and wooded environments, outdoor workers, outdoor sports enthusiasts, hunters and trappers	de la Cruz et al. 1984; Gese et al. 1997; Levesque et al. 1995; Cross and Penn 2000; Kaufman et al. 1997; CDC 2000b; CDC 2002b
Viral Diseases					
California serogroup viruses (Jamestown Canyon virus - JC, snowshoe hare virus-SSH) (genus *Bunyavirus*)	R: SSH: snowshoe hare (*Lepus americanus*) in some areas; other species equally or more important as maintenance and amplifying hosts, including rodents, carnivores, ungulates, ruffed grouse (*Bonasa umbellus*), chickens (*Gallus gallus domesticus*), dogs, horses and cattle; JC: white-tailed deer, other wild ungulates likely S: humans, occasionally horses V: SSH: mosquitoes (*Aedes, Culiseta, Culex* spp.); JC: mosquitoes (*Aedes* and *Anopheles* spp.)	SSH: infection reported from all 10 provinces, 3 territories; also northern U.S.; human illness in ON, QC, NB, NS JC: found in animals In NF, QC, ON, MB, stock, NWT; probably occurs throughout temperate North America; human cases rare - have occurred in ON, NWT Reportable: no	Expansion of range in Canada; milder winters increase overwinter survival of vector eggs; very high temperature will decrease adult mosquito survival; increased precipitation inundating tree-holes and other containers would improve larval habitat; rodent and small mammal reservoir population dynamics and health may be affected by changing climate; extreme winds might increase risk of infected mosquito drifting far beyond range; flooding increases mosquito habitat availability	Exposure linked to mosquitoes (outdoor activity, dawn and dusk); children and elderly especially vulnerable	Moore et al. 1993; Glass et al. 2000; Leighton 2000b
Dengue fever, dengue hemorrhagic fever (4 distinct dengue viruses)	R: humans, possibly birds V: mosquitoes (*Aedes aegypt* [exotic to Ontario], *A. albopictus* [in southeastern U.S.])	Globally epidemic; found in North America (southern U.S. and Mexico); human cases rare in Canada, all imported Reportable: no	Potential for expansion of range of competent vector into Canada; reservoir vector habitat includes areas with irrigated agriculture and stream drainages; more humid or more rain would promote mosquito habitat reproductive rates; migratory birds to the Great Lakes allowing vectors to become established; increased international air travel and immigration contribute to possible future dengue transmission in continental U.S., Imported cases in Canada	Travelers, children	WHO 2002a; Tsai 2000; Fayer 2000; Moore and Mitchell 1997; Mellor and Leake 2000; CDC 1996; CDC 2001a

Table A.2. Cont.

Disease (Agent)	Reservoir (R), Sentinel (S) and Vector (V)[1]	Current Disease Status[2]	Effects of Climate Variability and Change	Susceptible Populations	References[3]
Eastern equine encephalitis (EEE virus, genus *Alphavirus)*	R: passerine birds; also infects horses, some birds such as pheasants, quail, ostriches and emus, whooping crane (*Grus americana*) S: horses, imported birds (house sparrows [*Passer domesticus*], rock doves [*Columba livia*], etc.) V: endemic: mosquito (*Culiseta melanura*), bridge: mosquitoes (*Coquillettidia perturbans, Aedes sollicitans, A. vexans* and *Culex nigripalpus*)	13°C isotherm seems to limit occurrence of diesease; no recognized cases of human disease in Canada; occurs sporadically in several states bordering ON and QC; cases in horses have occurred in ON Reportable: no	Increased frequency of outbreaks if daily max above 30°C; expansion of range in Canada, milder winters increase overwinter survival of vector eggs, very high temperature will decrease adult mosquito survival; natural habitat of reservoir host and vector is swampy areas, increased precipitation would enlarge swamp habitat; outbreaks follow warm wet winter/dry hot summer, more humidity or more rain would promote mosquito reproductive rates; impact on avian reservoir possible, may affect transmission to humans	Extreme weather winds might increase risk of infected mosquito drifting far beyond range; flooding increases mosquito habitat availability; exposure linked to mosquitoes (outdoor activity, dawn and dusk)	Duncan et al. 1997; Reeves et al. 1994; Markoff 2000; Artsob et al. 1986; Calisher 1994; Moore et al. 1993; Sellers 1989; Sellers and Maarouf 1990; Freier 1993; Nasci and Moore 1998; Leighton 2000
Powassan encephalitis (POW virus, genus *Flavivirus)*	R: small mammals likely, marmot (*Marmota* spp.), snowshoe hare V: Ticks (*Ixodes marxi, I. cookei* and *D. andersoni*; recently, *I. Scapularis*)	Focally sporadic in ON and elsewhere in Canada where vectors are found Reportable: no	Northward expansion of range, milder winters increase overwinter survival of vector eggs, warmer weather may increase human exposure due to recreational activities in tick habitat, increased tick habitat if vegetation becomes more lush or dense, increased vertebrate host populations will increase number of infected ticks, by changes to vegetation, both host and vector habitats will be affected by precipitation - potential for epizootics when naive host population first infected	Exposure to ticks, grassy and wooded areas, outdoor workers, outdoor sports enthusiasts, dog owners, children, immuno-supressed, and elderly at elevated risk of clinical disease	CDC website 2001; Ebel et al. 1999; Tsai, 2000; Costero and Grayson 1996; Telford et al. 1997; Courtney et al. 2001; Artsob et al. 1986; Leighton 2000b
St. Louis encephalitis (SLE virus, genus *Flavivirus*)	R: birds V: Mosquitoes (*Culex pipiens* and other *Culex* species)	Widely distributed, Canada (MB) to Argentina, sporadic in Canada Reportable: no	Northward expansion of range, milder winters increase overwinter survival of vector eggs, very high temperature will decrease adult mosquito survivorship, vector larval development requires water in ditches, ponds, containers; population densities of *Cx. Pipiens* are highest in the dry season as water evaporates and organic concentration increases, impact on avian reservoir possible, may affect transmission to humans; extreme winds increase risk of infected mosquito drifting beyond range	Exposure linked to mosquitoes (outdoor activity, dawn and dusk), children and elderly especially vulnerable	Calisher 1994; Reeves et al. 1994; Shope 1980; Tsai 2000; Spence et al. 1977; Leighton 2000b

Table A.2. *Cont.*

Disease (Agent)	Reservoir (R), Sentinel (S) and Vector (V)[1]	Current Disease Status[2]	Effects of Climate Variability and Change	Susceptible Populations	References[3]
Western equine encephalitis (WEE virus, genus *Alphavirus*)	R: passerine birds, house sparrow, house finch (*Carpodacus mexicanus*); also ground squirrels (*Spermophilus* spp.), snakes, and leopard frog (*Rana pipiens*) S: horses, mosquitoes V: mosquitoes (*Culex tarsalis*, other *Aedes*, *Anopheles*, *Coquillettidia*, *Culex and Culesita* spp.)	13°C isotherm seems to limit occurrence of disease; southcentral Canada from Lake Superior to Rockies and BC Reportable: no	Expansion of range in Canada; milder winters increase overwinter survival of vector eggs; very high temperature will decrease adult mosquito survival; reservoir vector habitat is areas with irrigated agriculture and stream drainages; more humidity or more rain would promote mosquito habitat reproductive rates; less rain might result in greater areas under irrigation, also increasing mosquito habitat; impact on avian reservoir possible, may affect transmission to humans; extreme winds increase risk of infected mosquito drifting beyond range	Exposure linked to mosquitos (outdoor activity, dawn and dusk); children more vulnerable	Duncan et al 1997; Sellers and Maarouf 1988; Leighton 2000b
West Nile viral encephalitis (WN virus, genus *Flavivirus*)	R: birds, wide range of mammals S: birds from the Corvidae family, raptors, horses V: Mosquitoes (*Culex* spp.); *Cx. pipiens* likely the most important enzootic vector, *Cx. salinarius* possible bridge vector	Throughout the US; in birds in ON, QC, NS, MB, stock; human disease in QC, ON and AB, so far Reportable: federally	Expansion of range, elevated temperatures improve vector competence of *Cx. Pipiens*; vector larval development requires water in ditches, ponds, and containers, population densities of *Cx.pipiens* are highest in the dry season as water evaporates and organic concentration increases; extreme winds increase risk of infected mosquito drifting beyond range, climate change may alter disease ecology in reservoir passerine birds	Exposure linked to mosquitoes (outdoor activity, dawn and dusk), elderly, immuno-compromised more vulnerable	CDC and HC website 2002; Epstein 2001; Dohm et al. 2002; H. Artsob, pers comm. March 2002
Parasitic Diseases					
Babesiosis (*Babesia microti*)	R: White-footed mouse, meadow vole (*Microtus pennsylvanicus*) V: Black-legged tick	Coincides with range of black-legged tick; northeastern U.S., endemic; human cases very rare in Canada, all imported Reportable: no	Northward expansion of range; milder winters increase overwinter survival of vector eggs; warmer weather may increase human exposure due to recreational activities in tick habitat; increased tick habitat if vegetation becomes more lush or dense, increased vertebrate host populations will increase number of infected ticks; by changes to vegetation, both host and vector habitats will be affected by precipitation	Elderly, immuno-compromised, persons without spleens	Anderson et al. 1994; Varde et al 1998; Boustani and Gelfand 1996; Gadbaw et al 1989
Malaria (*Plasmodium falciparum, P. vivax, P. ovale and P. malariae*)	R: Humans are the intermediate host and reservoir S: humans V: Mosquitoes (*Anopheles* spp.)	Globally epidemic/endemic in most of the Tropics; sporadic cases transmitted in Texas eastward to New York State and eastern Seaboard, west to California and Oregon; almost all cases in Canada imported Reportable: federally, in ON	Northward expansion of range, accelerated development of sporogenic cycle within mosquito; natural habitat of vector is swampy areas, increased precipitation would enlarge swamp habitat; outbreaks follow warm wet winter/dry hot summer; more humidity or more rain would promote mosquito reproductive rates; extreme winds increase risk of infected mosquito drifting far beyond range	Mostly young children, travelers and immuno-compromised	Zucker 1996; CDC 1982; CDC 1986; CDC 1990; CDC 1991; CDC 1997b; ProMed mail posting Citing, Keystone, pers. Com; Bradley Et al. 2000; Layton Et al. 1995; Reiter 2000; R. Lindsay, pers. com; Fayer 2000 Health Canada 2002

Table A.3. *Potential impacts of climate change on direct zoonoses and rodentborne diseases of humans.*

Disease (Agent)	Reservoir (R), Vector (V)[1]	Current Disease Status[2]	Effects of Climate Variability and Change	Susceptible Populations	References[3]
Bacterial Diseases					
Anthrax *Bacillus anthracis*	R: Cattle carcasses, bison (*Bison bison*) in AB, other wild ungulates and contaminated soil	Sporadic in Canada, mostly in prairie provinces Reportable: in ON	Expansion of range possible due to increased activation of spores in warmer temperatures; Canadian epizootics have occurred in drought conditions following a wet spring; transmission decreases when cool wet weather returns; drought may pose increased risk by concentrating activated spores	Northern and north-western ON Aboriginal peoples, hunters and trappers, people who handle wildlife, outdoor workers (hydro, pipeline)	Coupland and Henderson 1996; Dragon et al. 1999; Gainer and Saunders 1989; Hugh-Jones 1999; Lusis and Smart 1996
Plague *Yersinia pestis*	R: Rats, prairie dog (*Cynomys ludovicianus*), Richardson's ground squirrel (*Spermophilus richardsonii*) V: Rat flea (*Xenopsylla cheopis*); clinical disease seen in cats (*Felis domesticus*), coyotes (*Canis latrans*)	Focally endemic in wild rodents in southern BC and AB; rare in humans Reportable: federally; in ON	Warmer temperatures would prolong transmission season, reduce impact of flea die-off in winter; possibly allowing for expansion of range; increased winter and spring precipitation enhances small mammal food resource productivity (plants and insects), leading to an increase in the abundance of plague hosts; moist climate conditions may promote flea survival and reproduction, enhancing plague transmission; increased rat-human contact if extreme weather causes loss of human shelter	Not likely a risk in ON Travelers to endemic foci in midwestern U.S. and elsewhere at risk, people in close contact with infected wildlife in endemic areas	Fowler and Henessey 1995; Lederberg et al. 1992; CDC 1995; CDC 1997a; Cheney 1998; Cully et al. 1997, 2000
Q Fever *Coxiella burnetii*	R: sheep (*Ovis aries*), goat (*Capra hircus*), cattle and wild ungulates V: rarely tickborne	Epidemic in sheep and possibly cattle in ON Reportable: in ON	Changes in small ruminant management practices that would result in increased risk of transmission from sheep, goats to humans - may be affected by warmer climate	People exposed to small ruminants (occupational exposure)	Levesque et al. 1995; Lang et al. 1991; Lang 1989; Maurin and Raoult 1999; Hawker et al. 1998; Wellock 1960

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[1] *Reservoir* refers to the animal or insect population in which infection is maintained. *Vector* refers to the arthropod that transmits the infection between animals and from animals to people.

[2] *Federally reportable* diseases are nationally notifiable (all cases must be reported to Health Canada). Some provinces conduct surveillance of additional diseases. Diseases that are notifiable in Ontario, but not necessarily in other provinces, are listed as *reportable in ON*.

[3] Full references are listed in Part B of this report.

Table A.3. Cont.

Disease (Agent)	Reservoir (R), Vector (V)^1	Current Disease Status^2	Effects of Climate Variability and Change	Susceptible Populations	References^3
Tuberculosis, M. avium complex (MAC) *M. bovis, M. avium, M. intracellulare, M. tuberculosis*	R: wild ungulates, birds; also occurs in other animals	Resurgence globally, but sporadic in Canada, human cases usually imported; infected domesticated elk, possibly wild ungulates reported in ON Reportable: in ON; federally	Warmer climate may allow cattle grazing and other human activity in areas inhabited by *M. bovis* infected wildlife; major weather disasters internationally may increase risk of imported human disease, in particular multi-drug resistant tuberculosis	Sporadic cases may occur due to *M. bovis* through exposure to infected wildlife carcass; MTB and MAC affect people with HIV/AIDS	CDC 2002; Coisivi et al. 1998; Haas 2000; WHO 2000; Grondin 2002; WHO 2002b; Dankner et al. 1993; Health Canada 2002
Tularemia Type B *Franciscella tularensis*	R: beaver (*Castor canadensis*) and muskrat (*Ondatra zibethicus*), wild rabbits, hares	Very common in beaver, muskrat; less common in rabbits across southern Canada; hares much less likely to carry the organism; disease is extremely rare, sporadic in ON Reportable: in ON	Most cases occur from direct transmission from infected animals; can be waterborne, but very rarely; this form is not usually vectorborne; by changes to vegetation, host (and vector) habitats will be affected by precipitation; potential for epizootics when naive host population first infected; possible waterborne outbreaks if beaver/muskrat population heavily infected	Northern Ontarians, Aboriginal peoples, hunters and trappers, people who handle wildlife, people exposed to infested waters	de la Cruz 1984; Gese et al. 1997; Levesque et al. 1995; Cross and Penn 2000; Kaufman et al. 1997; CDC 2000b; CDC 2002
Viral Diseases					
Rabies Rabies virus	R: Skunk (*Mephitis mephitis*), fox (*Vulpes vulpes*), bats New strain could become endemic in raccoons (*Procyon lotor*), can affect all mammals: ungulates, canids, felines, etc.	Endemic with epizootics in wildlife; human disease is very rare in Canada, recent cases linked to bat exposure Reportable: federally; in ON	Warmer summers may change range of reservoir species, prolong foraging season for wildlife; these factors may bring rabies outbreaks in previously quiescent areas, or new strains into naive wildlife populations (e.g., raccoon rabies); bats may prefer warmer climate; changes in humidity and precipitation may alter vegetation, and change wildlife habitat	Suburban areas where wildlife thrive; raccoon strain may pose additional risk if epidemic spreads; cottagers, campers, potential exposure to bat bites, unvaccinated pets a potential public health threat	Duncan et al. 1997; Bleck and Rupprecht 2000; Turgeon et al. 2000; Varughese 2000; Nunan et al. 2002; MacInnes et al. 2001; Hanlon and Rupprecht 1998; MacInnes et al. 2001

Table A.3. Cont.

Disease (Agent)	Reservoir (R), Vector (V)[1]	Current Disease Status[2]	Effects of Climate Variability and Change	Susceptible Populations	References[3]
Hantavirus pulmonary syndrome Hanta viruses	R: Deer mouse (*Peromyscus oreas*), white-footed mouse, other rodents	Sporadic occurrence in the U.S.; human cases rare in Canada Reportable: federally	Warmer summers may increase rodent population; mild winters reduce mortality; expansion of range; increased precipitation increases rodent population densities in Hanta-endemic areas; inclement weather may force rodents indoors increasing exposure; major weather disasters and loss of shelter may contribute to increased exposure	People exposed to rodent droppings – occupational (cleaners, etc.) or cottagers, campers, etc.; workers housed in temporary camps (hydroline, pipeline, etc.); may occur in urban areas also	Hjelle and Glass 2000; Gubler et al. 2001; Steere 2000; Health Canada 2000; CDC 2000b; Wenzel 1994; Stone 1993; Glass et al. 2000
Influenza Influenza virus, type A	R: aquatic birds, ducks, geese, pigs, poultry	Seasonally epidemic in Canada Reportable: federally; in ON	Pandemics have generally begun in Asia; global warming may result in new locations being more vulnerable to epizootics; changes in global climate may alter ecology of current animal reservoirs, contributing to outbreak of a new pandemic strain	Outdoor poultry and fowl markets may be potential source	Swayne 2000; Treanor 2000; Macey et al. 2002; Webster 1998; Karasin et al. 2000; Webster et al. 1981; Geraci et al. 1982; van Campen and Early 2001; Chan 2002

Table A.4. *Potential impacts of climate change on foodborne diseases of humans.*

Disease (Agent)	Reservoir[1]	Current Disease Status[2]	Effects of Climate Variability and Change	Susceptible Populations	References[3]
Bacterial Diseases					
Brucellosis *(Brucella aborta, B. melitensis, B. suis, B. canis)*	R: wild ruminants, bison *(Bison bison)*, seals Disease occurs in cattle, dogs *(Canis domesticus)*, other mammals	Focally endemic in wild ungulates; human disease is rare in Canada Reportable: federally; in ON	Warmer climate may allow cattle grazing in areas inhabited by infected wildlife; more favourable conditions, especially in sub-Arctic, may predispose to new strains; changes in sea levels may affect disease levels in marine animals; may have implications for sub-Arctic	Aboriginal peoples, hunters	Ferguson 1997, Forbes et al. 2000; Forbes and Tessaro 1996; Meyer and Meagher 1995; Young 2000
Campylobacteriosis *(Campylobacter fetus* subsp. jejuni, also subsp. fetus, intestinalis)	R: Cattle, sheep *(Ovis aries)*, goats *(Capra hircus)*, dogs, poultry, pigs *(Sus domesticus)*, wild birds, cats *(Felis domesticus)*, rodents, flies	Endemic Reportable: federally; in ON	Increased risk of contamination and proliferation of organisms during meat processing, transportation and storage; of proliferation and transmission in wildlife; of human exposure related to prolonged barbecue and camping season; known to be waterborne; increased agricultural runoff and contamination of surface water by wildlife increases human exposure and risk of transmission	All Ontarians, especially the young, elderly and the sick	Isaacs et al. 1998; Gaudreau and Gilbert 1998; Ellis et al. 1995; Irving 2001
Clostridium botulinum **food poisoning** (Types A, B, E and F)	R: ubiquitous anaerobic environmental organism; affects waterfowl, fish	Type E recently epidemic in waterfowl and fish in Lakes Erie and Ontario Human disease sporadic, linked to improperly preserved game meats Reportable: federally, in ON	The dynamics of the disease in fish and birds in the Great Lakes are not clear, but seem to be related to some warming trends; the implications for human health risk at this time are not clear	Native peoples subsisting on traditional diet; people who eat home-canned or artisan foods, in particular fish	Health Canada 1999

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[1] *Reservoir* refers to the animal or insect population in which infection is maintained.

[2] *Federally reportable* diseases are nationally notifiable (all cases must be reported to Health Canada). Some provinces conduct surveillance of additional diseases. Diseases that are notifiable in Ontario, but not necessarily in other provinces, are listed as *reportable in ON*.

[3] Full references are listed in Part B of this report.

Table A.4. Cont.

Disease (Agent)	Reservoir[1]	Current Disease Status[2]	Effects of Climate Variability and Change	Susceptible Populations	References[3]
***Clostridium perfringens* food poisoning** *(Clostridium perfringens,* Type A)	R: Ubiquitous environmental organism; many domestic and wild animals	Sporadic Reportable: no	Increased risk of contamination and proliferation of organisms during meat processing, transportation and storage; of proliferation and transmission in wildlife; of human exposure related to prolonged barbecue and camping season; outbreaks of avian botulism generally occur on water bodies with little or no outflow; grow best during periods of hot dry weather; extreme hot dry weather will promote the growth of the organism	All Ontarians, especially the young, elderly and sick	CDC website 2002a
Listeriosis *(Listeria monocytogenes)*	R: ubiquitous environmental organism; asymptomatic carriers: many mammals including humans, birds, fish, and crustaceans	Rare Reportable: no	The risk of listeriosis in Ontario is related to food choices and underlying susceptibility; changes in climate may increase the potential for contamination of milk and meats with pathogen, but is unlikely to affect overall risk of disease in people	The elderly, newborns, and those with compromised immune function; risk to fetus during pregnancy	HC-PPHB 2002a
Salmonellosis *(Salmonella* spp., including multi-drug-resistant *Salmonella typhimurium* Dt104)	R: Mammals, reptiles, birds	Endemic Sporadic epidemics Reportable: federally; in ON	Increased risk of contamination and proliferation of organisms during meat processing, transportation and storage; of human exposure related to prolonged barbecue and camping season	All Ontarians, especially the young, elderly and sick	Buxton et al. 1999; Isaacs et al. 1998; Khakhria et al. 1997; Ahmed et al. 2000; Irving 2001; Buck and Werner 1998; Hosek et al. 1997; Daoust et al. 2000; Letellier et al. 1999; Mikaelian et al. 1997; van Donkersgoet et al. 1999; Ackers et al. 2000
Shigellosis *(Shigella* spp.)	R: Humans also dogs, horses, bats, rattlesnakes (*Crotalus* spp.), captive monkeys	Focally endemic with sporadic epidemics across Canada Reportable: federally; in ON	Increased survival of organism in warmer temperatures; increased transmission in surface water after high precipitation event; flooding will increase risk from human sewage; potential for outbreaks in favoured travel destinations	Travellers, Aboriginal communities	Irving 2001; Crowe et al. 1999; Rosenberg et al. 1997

Table A.4. Cont.

Disease (Agent)	Reservoir[1]	Current Disease Status[2]	Effects of Climate Variability and Change	Susceptible Populations	References[3]
Staphylococcal food poisoning *(Staphylococcus aureus)*	R: Domestic and wild animals	Sporadic occurrence across Canada; pathogen is ubiquitous Reportable: in ON	Increased risk of contamination and proliferation of organism during meat processing, transportation and storage; of human exposure related to prolonged barbecue and camping season		CDC website 2002
VTEC and colibacillosis *(Escherichia coli, E. coli* O157)	R: Many domestic animals; O157:H7 in cattle, especially dairy calves	Endemic with sporadic epidemics across Canada Reportable: federally; in ON	Increased risk of contamination and proliferation of organism during meat processing, transportation and storage; of human exposure related to prolonged barbecue and camping season; increased risk with increased agricultural runoff from high rains; flooding will increase risk	All Ontarians, especially the young, elderly and the sick	Van Donkersgoed et al. 1999; Khakhria et al. 1997; Letellier et al. 1999; Mikaelian et al. 1997; O'Connor 2002
Infections and Intoxications From Fish and Seafood					
Ciguatera poisoning paralytic (PSP), amnesic (ASP), and diarrheic (DSP) shellfish poisonings *Vibrio parahaemolyticus* , various environmental and fecal pathogens	Many of these linked to dinoflagellate or cyanobacterial (blue-green algae) blooms	Sporadic, rare in ON	Algal blooms increasing in frequency, may be related to climate change; ecology of estuaries and oceans will likely be profoundly affected by climate change with implications for algal bloom frequency and distribution; Ontarians who consume fish and shell fish are at risk of illness related to these blooms	Those who eat fish and seafood, travellers	Tester 1994
Viral Diseases					
Enteric viruses (Norwalk, calicivirus, etc.)	R: humans	Endemic Reportable: no	Like other viruses which are not zoonotic (or primarily reside in humans), climate change is unlikely to affect disease transmission through food	Children, the elderly, immunocompromised	Health Canada 1999, 2002b
Hepatitis A virus	R: humans	Sporadic outbreaks, travel-related cases, sexually transmitted Reportable: federally; in ON	Like other viruses that are not zoonotic (or primarily reside in humans), climate change is unlikely to affect disease transmission through food		Feinstone and Gust 2000, HC-PPHB 2000

Table A.5. *Potential impacts of climate change on waterborne diseases of humans.*

Disease	Reservoir[1]	Current Disease Status[2]	Effects of Climate Variability and Change	References[3]
Bacterial Diseases				
Cholera (*Vibrio cholerae*)	R: marine dinoflagellates, humans	Pandemic; recent spread to Americas; few imported cases reported annually in Canada (sometimes from ON); few cases have no travel history Reportable: federally; in ON	Outbreaks elsewhere almost always linked to warm weather; climate change may affect the worldwide distribution of cholera, altering the risk of the disease to Ontarians traveling abroad; the potential for the disease to become established in Ontario is minimal, since Ontario's marine shores will remain too cold to support the vibrio; a sustained warming of Great Lakes temperatures, and contamination of these waters with cholera, could result in the focal establishment of the disease	Seas and Gotuzzo, 2000 Health Canada 2002a, WHO 2001
Enteric bacteria See Table A.4.	R: See Table A.4.	Epidemic across Canada and also sporadic; some are endemic Reportable: see Table A.4	Increased survival of organism in warmer temperatures; increased human exposure by exposure to contaminated water (swimming, camping, etc.); increased transmission in surface water after high precipitation event; increased risk with increased agricultural runoff from high rains; flooding will increase risk from human sewage, sewer overflows	See Table A.4
Legionellosis *Legionella pneumophila*, other spp.	R: relatively common environmental pathogen, prefers stagnant water, found in cooling systems, towers, ponds	Sporadic but not rare in ON Reportable: federally; in ON	A sustained warmer climate and more frequent air quality problems in Ontario may increase in the risk of legionellosis; persons already at increased risk of the disease (the elderly and those with chronic lung disease) will be required to spend even more time in air-conditioned environments, or have increased need of various breathing apparatus such as oxygen masks; the impact of climate change on other sources of this disease is not known at present	Yu 2000

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[1] *Reservoir* refers to the animal or insect population in which infection is maintained.

[2] *Federally reportable* diseases are nationally notifiable (all cases must be reported to Health Canada). Some provinces conduct surveillance of additional diseases. Diseases that are notifiable in Ontario, but not necessarily in other provinces, are listed as *reportable in ON*.

[3] Full references are listed in part B of this report.

Table A.5. Cont.

Disease	Reservoir[1]	Current Disease Status[2]	Effects of Climate Variability and Change	References[3]
Leptospirosis *Leptospira interrogans*, other spp.	R: Rodents, wild mammals (lynx [*Lynx lynx*], raccoon [*Procyon lotor*]), horses, (*Equus equus*), dogs, cattle, pigs (different serovars)	Epidemic and also sporadic; rare in Canada. Reportable: no	Increased risk of organism survival if water temperatures increased for longer periods; increased human exposure by exposure to contaminated water (swimming, camping, etc); increased risk with increased runoff containing infected urine and contamination of surface water, both wild and domestic animal sources; flooding will increase risk	Duncan et al. 1997; Levesque et al. 1995; Hrinivich and Prescott 1997, 1999; Kalin et al. 1999; Labell et al 2000; Mikaelian et al. 1997; Richardson et al. 1995; Shapiro et al. 1999
Shigellosis *Shigella* spp.	R: Dogs, horses, bats, rattlesnakes, captive monkeys	Epidemic and endemic across Canada Reportable: federally; in ON	Increased survival of organism in warmer temperatures; increased transmission in surface water after high precipitation event; flooding will increase risk from human sewage	Irving 2001; Crowe et al. 1999; Rosenberg et al. 1997
Parasitic Diseases				
Cryptosporidiosis *Cryptosporidium parvum*, other spp.	R: Humans, calves, lambs, other domestic animals; infectious in 80 animal species	Epidemic across Canada and also sporadic Reportable: in ON	Increased persistence of organism if water temperatures increase for longer periods; increased risk with increased agricultural runoff from high rains; flooding will increase risk	MacKenzie et al. 1994; Meinhardt et al. 1996; Goldstein et al. 1996; Rose 1997
Giardiasis *Giardia lamblia*	R: Domestic and wild animals, including beaver (*Castor canadensis*), dogs, pigs	Epidemic and sporadic across Canada Reportable: federally; in ON	Increased risk if water temperatures increase for longer periods; increased risk with increased agricultural runoff from high rains; flooding will increase risk	Duncan et al. 1998; Kramer et al. 1996
Toxoplasmosis *Toxoplasma gondii*	R: Sheep, pigs, wild and domestic cats, rodents, birds	Epidemic and also sporadic; rare in Canada. Reportable: no	Increased persistence of organism in the environment with warmer temperatures; increased risk with increased runoff from high rains and contamination by both wild fecal material and manure from livestock; flooding will increase risk	Duncan et al. 1998; den Hollander and Notenboom 1996

Executive Summary: Diseases in Animals

Diseases in animals are important for the animals that suffer from them and for the people who raise them, eat them, and/or share environmental spaces with them. Many of the bacteria, viruses and parasites that infect animals - such as rabies, West Nile virus, *Escherichia coli* O157, and some intestinal parasites of dogs and cats - can be transmitted to people. Others - such as some fungi or environmental contaminants - may show up in both people and animals because we share common environments. Still other agents may have sufficient impact on agricultural animals that the livelihoods of farmers and rural communities suffer, often severely (for example, the outbreak of foot and mouth disease in the United Kingdom in 2001). Whatever their effects on the animals themselves, animal disease outbreaks may serve as sentinels or early warnings for ecosystem and human problems.

Disease agents are transmitted in a variety of ways. Some diseases, such as rabies, brucellosis and tuberculosis, are transmitted among animals and from animals to people more or less directly. Other agents can be transmitted through insect vectors (Lyme disease, West Nile virus), through water (*E. Coli* 0157:H7, *Campylobacter fetus*), through food such as meat (*Salmonella* spp. to people, *Trichinella* spp. among animals and to people), or through general environmental contamination (parasitic larvae from dogs and cats, Q fever). Some disease agents, such as fungi and the toxins they produce, live in the environment, from which they affect many species. Most of these diseases are in some way affected by weather patterns and, more generally, by climate. In the decades since World War II, many new animal diseases and livestock and environmental management practices have evolved and adapted to the particular set of climatic and environmental conditions in Ontario. Increasingly, both averages and extreme events are taking us outside those conditions, which are reflected in changes in disease patterns. If we wish to keep our human and animal populations healthy, our disease and environmental management practices must continue to change as well.

Changing rainfall patterns and milder temperatures affect the survival and amplification of insect vectors (ticks, mosquitoes) and allow diseases previously considered exotic or rare (West Nile, leishmaniasis) to invade and survive in Ontario. Milder winters also affect disease dynamics by changing the population dynamics of host animals and/or changing the ranges of some animals, such as raccoons, deer and opossum. A wide variety of fungi and their toxins are already present in Ontario soils and crops; they will likely increase in profile and importance where drought damages crops and wet, mild winters encourage fungal growth. Although milder winters may reduce the occurrence of some diseases, such as pneumonia in adult cattle, many more diseases are likely to become more common. Indeed, many diseases of young livestock, such as pneumonia and diarrhea, as well as diseases in farmed fish, are more affected by rapid changes in temperature and moisture than by averages. Milder winters affect parasite survival in and on animals and could contribute to an increase in parasite loads in livestock, dogs, and cats. Also, bacterial and parasitic contamination could occur more frequently in watersheds that supply drinking water. Heavy rainfall - or periods of drought followed by heavy rains - flush contamination through the watersheds, resulting in a greater probability of outbreaks such as those seen in Victoria (toxoplasmosis) and Walkerton (*E. coli* and campylobacteriosis). Although this is of greatest concern for people, farmed fish and livestock are also affected by contaminated runoff.

Aquatic animals, and birds relying on them, are highly vulnerable to climate change. Systemic interactions among multiple species, infectious agents, water temperatures and runoff from the land respond more quickly and pass through critical thresholds between ecosystemic patterns of organization more dramatically than in most land-based systems. Mitigation actions will be crucial for long-term survival of many species in the Great Lakes, smaller Ontario lakes, and the James Bay/Hudson's Bay area. In the latter case, the livelihoods of people in the north will be affected.

More detailed information on the effects of climate change on diseases of animals is listed in tables A.6 to A.9.

With the exception of catastrophic events such as ice storms and floods, most of the effects of climate on animal (and human) diseases are mediated through complex social and ecological interactions and feedback loops. Climate trends will change but will be manifested in a variety of local weather patterns, which will have differing effects depending on local social and ecological conditions (urbanization, agriculture, landscape structures). The specific diseases we are concerned with vary over time and across the province and the country. Responses to possible threats will therefore need to take an adaptive approach and will require involvement from the general public; a diversity of government departments at local, provincial and national levels; and a wide variety of scientific scholarly disciplines. We believe that the best way to achieve this is through a combination of national commitment and steering committees, research and surveillance networks, and strong local response capability. The basic structure of the steering committee and multi-institutional, multi-disciplinary network system has already been put into place through the Health Canada Climate Change and Health Office (HC-CCHO 2002, electronic access) and related initiatives through the Canadian Climate Impacts and Adaptation Research Network (C-CAIRN 2002, electronic access). The success of these initiatives will require strong logistical and financial support from several levels of government, across government departments, and from universities. Ontario government departments need a strong provincial presence in these networks and monitoring systems, given the importance of Ontario as a focal point for many emerging climate-related diseases. Initiatives to work with communities so they can play a more active role in the networks and monitoring systems and use the information from those systems to develop locally adaptive strategies for watershed management, land zoning, and public health education are also needed.

Table A.6. *Potential effects of climate change on the health of domestic animals and wildlife.*

Climate Parameter	Potential Direct Effects	Potential Indirect Effects
Increased temperatures (particularly night-time lows during winter and at northern latitudes)	- Morbidity and mortality associated with thermal stress	- Alterations in habitat - Altered phenology/synchronicity of life cycle events - Alterations in incidence and distribution of vector-borne and parasitic diseases - Alterations in quality/quantity of food supply
Altered amount and pattern of precipitation		- Alterations in habitat - Alterations in water quality - Increased risk for waterborne disease with flooding - Alterations in incidence and distribution of vectorborne and parasitic diseases - Alterations in quality/quantity of food supply
Altered patterns of extreme weather events (convective storms, floods, ice storms, tornadoes)	- Direct mortality	- Increased risk of power outages and effects on housed livestock - Increased risk for waterborne disease with flooding - Altered potential for airborne pathogen distribution - Altered airborne vector dispersal patterns
Stratospheric ozone depletion and increased ultraviolet radiation	- Increased incidence of ocular squamous cell carcinoma - Effects on immune function	- UV-mediated effects on free-living waterborne parasite stages

Table A.7. *Potential effects of climate change on non-vectorborne infectious diseases of animals.*

Disease Agent	Species at Risk for Clinical Disease	Current Disease Status[1]	Maintenance Reservoir	Transmission	Effects of Climate Variability and Change	Reference[2]
Bacterial Diseases						
Anthrax (*Bacillus anthracis*)	Wild and domestic animals (especially herbivores), humans	Sporadic occurrence in Canada; OIE LIST B	Fecal shedding from infected herbivores, infected soil	Waterborne, foodborne mechanical, inhalation	Spores very resistant; improved environmental conditions for dissemination and concentration of spores	Gates et al. 2001; CFIA 2001a
Campylobacteriosis (*Campylobacter jejuni*)	Dogs (*Canis domesticus*), cats (*Felis domesticus*), humans	Sporadic and epidemic occurrence in Canada; OIE LIST A	Fecal shedding from livestock	Waterborne, foodborne	Increased potential for fecal contamination of water; flooding may increase risk for waterborne diseases	Harmon et al. 2000; Radostits et al. 2000
E. coli enteritis (*Escherichia coli*)	Domestic livestock (calves, lambs (*Ovis aries*), kids (*Capra hircus*), pigs (*Sus domesticus*), foals (*Equus equus*), humans	Reportable: federally, in ON; sporadic occurrence in Canada; OIE LIST A	Fecal shedding from livestock	Waterborne, foodborne	Improved environmental conditions for proliferation of organism; stress precipitates disease; flooding may increase risk for waterborne diseases	Harmon et al. 2000; Radostits et al. 2000
Leptospirosis (*Leptospira* spp.)	Cattle, horses, dogs, swine; humans may be incidental hosts for some serovars	Sporadic disease in Ontario; infection in animals (endemic in raccoons [*Procyon lotor*]); OIE LIST B	Many, serovar-specific, including raccoon, skunk (*Mephitis mephitis*), opossum (*Didelphis virginiana*), mice, rats, fox (*Vulpes vulpes*), squirrels, muskrat (*Ondatra zibethicus*), etc.	Waterborne	Improved environmental conditions for proliferation of organism; flooding may increase risk for waterborne diseases	Parmenter et al. 1999; Enscore et al. 2002; Bolin 2000
Salmonellosis (*Salmonella* spp.)	Domestic livestock, dogs, cats, humans	Reportable: federally, OIE LIST A; *Typhurium, Galinarum* - endemic occurrence in Canada (wild birds)	Fecal shedding from livestock and other reservoirs, including wild animals and birds	Waterborne, foodborne	Improved environmental conditions for proliferation of organism; increased potential for fecal contamination of water; flooding may increase risk for waterborne diseases	Harmon et al. 2000; Radostits et al. 2000

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[1] *OIE listing* refers to the Office Internationale des Epizooties classification of diseases. In Canada, all *OIE list A* diseases are nationally reportable. The *OIE list B* diseases are of national interest but are not notifiable. The Canadian Food Inspection Agency collects information on OIE list A and list B diseases.

[2] Full references are listed in Part C of this report.

Table A.7. Cont.

Disease Agent	Species at Risk for Clinical Disease	Current Disease Status[1]	Maintenance Reservoir	Transmission	Effects of Climate Variability and Change	Reference[2]
Tuberculosis (*Mycobacterium bovis*)	Wide host range, domestic animals, wildlife, humans	Occurs across the U.S.; sporadic and epidemic cases in Canada; reportable: federally, in ON	Free-living and farmed deer	Inhalation, foodborne	Alterations in habitat and range or abundance of reservoir hosts; maximum survival of organism in environment in cold, damp conditions	CFIA 2001a; Radostits et al. 2000; Acha and Szfres 2001; Clifton-Hadley et al. 2001
Tularemia Type B (*Francisella tularensis palaearctica*)	Muskrat, beaver (*Castor canadensis*), hare, humans (mild)	Canada and northern U.S.; OIE LIST A	Muskrat, beaver, snowshoe hare (*Lepus americanus*)	Direct contact, inhalation, waterborne, foodborne	Habitat loss, stress precipitating disease outbreaks; increased potential for contamination of water	Markowitz et al. 1985; Gese et al. 1997; Levesque et al. 1995
Yersiniosis (*Yersinia enterocolitica, Y. pseudotuberculosis*)	Rarely cattle, sheep, pigs, goats, humans, farmed deer	Sporadic distribution of disease across Canada (wild birds and deer) with epidemic outbreaks; single most common cause of death in beavers	Fecal shedding from livestock and other reservoirs including wild animals, birds	Waterborne, foodborne	Environmental stress precipitates disease; increased potential for contamination of water; flooding may increase risk for waterborne diseases	Harmon et al. 2000; Radostits et al. 2000
Viral Diseases						
Distemper (Morbilliviruses)	Dog, raccoon, coyote (*Canis latrans*), wolf (*C. lupus*), fox, mustelids, harbour seal, harp seal	Endemic in ON	Domestic and wild carnivores including raccoons and skunks, marine mammals	Aerosol or direct	Alterations in habitat and range or abundance of reservoir hosts	Schubert et al. 1998
Influenza A (Orthomyxovirus)	Pigs, domestic poultry, water fowl, humans, horses, harbour seals	Reportable: in ON; endemic in Canada	Aquatic birds (waterfowl, shorebirds)	Aerosol or direct	Alterations in habitat and range or abundance of reservoir hosts increases risk of interspecies transmission	Chan 2002; Geraci et al 1982; Halvorson et al. 1985; Karasin 2000b; Karasin 2000a, Swayne 2000; Webster et al. 1981; van Campen and Early 2001

Table A.7. Cont.

Disease Agent	Species at Risk for Clinical Disease	Current Disease Status[1]	Maintenance Reservoir	Transmission	Effects of Climate Variability and Change	Reference[3]
Newcastle disease (Paramyxovirus)	Domestic poultry, cormorant (*Phalacrocorax auritus*)	Sporadic distribution: OIE LIST A	Wild waterfowl (cormorants, gulls)	Aerosol, cloacal excretion	Alterations in habitat and range or abundance of reservoir hosts increases risk of interspecies transmission	Wobeser 1997; Banerjee et al. 1994; Glaser et al. 1999
Rabies (Lyssavirus)	Warm-blooded animals	Focal epidemics: OIE LIST B	Fox, striped skunk (*Mephitis mephitis*), raccoon, bats	Direct	Alterations in habitat and range or abundance of reservoir hosts	MacInnes et al. 2001; Mitchell et al. 1999; Christensen et al. 1993; Rupprecht 2001
Fungal Diseases						
Blastomycosis (*Blastomyces dermatitidis*)	Dogs, humans, wolves, foxes	Endemic range in the eastern U.S. and Canada and in northern ON (Muskoka, Kenora)	Soil	Free-living soil phase; airborne inhalation of spores	Improved environmental conditions for proliferation of organism	Legendre 1998; McEwen and Hulland 1984
Cryptococcosis (*Cryptococcus neoformans*)	Cats, dogs, humans, wild mammals	Epidemic in western Canada (Vancouver Island 2002)	Pigeon droppings	Free-living phase; airborne inhalation of spores	Improved environmental conditions for proliferation of organism	Jacobs and Medleau 1998; Health Canada 2002
Histoplasmosis (*Histoplasma capsulatum*)	Cats, dogs, humans	Endemic in eastern North America; occasional	Soil	Free-living soil phase; airborne inhalation of spores	Improved environmental conditions for proliferation of organism	Wolf 1998
Other						
Chronic wasting disease (Prion disease)	Elk (*Cervus elaphus*), mule deer (*Odocoileus hemionus*), white-tailed deer (*O. Virginianus*	Reportable: federally and in ON (AB, Toronto); sporadic western to eastern distribution in U.S. and Canada	Rocky Mountain elk, mule deer, white-tailed deer	Unknown	Alterations in habitat and range or abundance of reservoir hosts	Wisconsin DNR 2003, electronic access; Williams et al. 2001; Holland 2002

Table A.8. *Potential effects of climate change on vectorborne diseases of animals.*

Disease (Agent)	Species at Risk for Clinical Disease	Current Disease Status[1]	Vector (v) and Natural Maintenance Hosts (H)	Effects of Climate Variability and Change	Reference[2]
Bacterial Diseases					
Canine monocytic ehrlichiosis (*Ehrlichia canis*)	Canids (coyote (*Canis latrans*), foxes (*Vulpes* spp.), dogs (*Canis domesticus*)	Worldwide; reportable: no	V: Brown dog tick (*Rhipicephalus sanguineus*) R: wild canids	Altered geographic distribution of vector species, impact on susceptible wild animal host populations, affecting disease dynamics, wildlife populations and domestic animal exposure	Neer 1998
Equine granulocytic ehrlichiosis (*Ehrlichia equi, Ehrlichia risticii*)	Horses (*Equus equus*), humans (*E. equi* genogroup)	Reportable: federally; sporadic in North America	V: Black-legged tick (*Ixodes scapularis*), western black-legged tick (*I. pacificus*) H: Horses	Altered geographic distribution of vector species affecting disease dynamics and equine exposure; possible expansion of range; milder winters increase overwinter survival of vector eggs; increased tick habitat if vegetation becomes more lush or dense; by changes to vegetation, both host and vector habitats will be affected by precipitation - potential for epizootics when naïve host population first infected	Drebot et al. 2001; Neer 1998
Lyme disease (*Borrelia burgdorferi*)	Humans, dogs, cats (*Felis domesticus*)	Endemic in southeastern ON; reported in 48 states, mostly eastern coastal states, upper Midwest and northern California; reportable in ON	V: Black-legged tick, western black-legged tick R: Larvae and nymphs: rodents, especially white-footed mouse (*Peromyscus leucopus*), small mammals, birds; Adults: deer, larger mammals	Altered geographic distribution of vector species, impact on susceptible animal host populations, affecting disease dynamics; possibly increased suitability of some habitats due to increased humidity; northward expansion of range; milder winters increase overwinter survival of vector eggs; tick habitat includes lush or dense vegetation; increased vertebrate host populations will increase number of infected ticks	Barker and Lindsay 2000; Ostfeld and Keesing 2000b; Lindsay et al. 1999; Lindsay et al. 1995; Shih et al. 1995
Rocky Mountain spotted fever (*Rickettsia rickettsii*)	Humans, dogs	Occurs throughout the contiguous U.S.; sporadic, rare in Ontario; reportable: western Canada	V: Dog tick (*Dermacentor variabilis*) (eastern N. America), wood tick (*D. andersonii*) (western N. America) R: Rodents, small mammals, possibly raccoon (*Procyon lotor*), foxes, opossums (*Didelphis virginiana*)	Northward expansion of range; milder winters increase overwinter survival of vector eggs and have other impacts on tick ecology, affecting transmission	Greene and Breitschwerdt 1998
Tularemia Type A (*Francisella tularensis tularensis*)	Rabbits, humans	Occurs across the U.S. and southern Canada; highly virulent; reportable: no	V: Dog tick, wood tick, and others, biting flies H: Cottontail rabbit (*Sylvilagus floridanus*), jack rabbits (*Lepus townsendii*)	Altered geographic distribution of vector species; tick and deerfly vector ecology will be affected by increased temperature, resulting in potentially greater levels of infection in vectors, prolonged transmission season, and reduced winter die-off; by changes to vegetation, both host and vector habitats will be affected by precipitation, potential for epizootics when naïve host population first infected	Markowitz et al. 1985; Gese et al. 1997; Levesque et al. 1995

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[1] *OIE listing* refers to the Office Internationale des Epizooties classification of diseases. In Canada, all *OIE list A* diseases are nationally reportable. The *OIE list B* diseases are of national interest but are not notifiable. The Canadian Food Inspection Agency collects information on OIE list A and list B diseases.

[2] Full references are listed in Part C of this report.

Table A.8. Cont.

Disease (Agent)	Species at Risk for Clinical Disease	Current Disease Status[1]	Vector (v) and Natural Maintenance Hosts (H)	Effects of Climate Variability and Change	Reference[2]
Parasitic Diseases					
Canine babesiosis *(Babesia canis, B. Gibsoni)*	Dogs, rarely humans	U.S., particularly Gulf Coast states	V: Brown dog tick	Altered geographic distribution of vector species, impact on susceptible wild animal host populations, affecting disease dynamics, wildlife populations and domestic animal exposure	Taboada 1998
Heartworm (*Dirofilaria immitis*)	Dogs, cats, rarely humans	Widespread in U.S., southern ON, QC, MB, Okanagan Valley of BC	V: Mosquitoes (*Aedes vexans, Ae. taemorhynchus , Ae. solicitans, Cx. salinarius, Cx. quinquifasciatus*, and *Ae. quadrimaculatus*) R: Coyote (*Canis latrans*), foxes, wolf (*C. lupus*)	Altered geographic distribution of vector species, including northward expansion of range; enhanced vector competence due to warm temperatures, longer transmission season possible, potential for new vector species and new host species (cats, for example)	Slocombe 1990; Slocombe and Villeneuve 1993
Leishmaniasis (*Leishmania infantum*)	Dogs, humans	21 states, sporadic in ON, in foxhounds	V: Sand flies (*Phlebotomus, Lutzomyia*); direct R: Wild and domestic dogs	Impacts of climate on sand flies and on transmission of leishmaniasis in ON are not known at present	Slapendel and Ferrer 1998; Irwin 2002; Gaskin et al 2002
Potomac horse fever (*Ehrlichia risticii*)	Horses	Diagnosed in ON, sporadic; occurs across the U.S.	V: Trematodes in family Lecithodendriidae H: bats, birds 1° IH: pleurocerid snails 2nd IH: aquatic insects	Increased availability of wet habitat for vectors and intermediate host	Madigan and Pusterla 2000
Viral Diseases					
Bluetongue (Orbivirus)	Sheep (*Ovis aries*), domestic and free-living deer	Southern and western U.S., sporadic incursions into southern B.C and Alberta (Cypress Hills); OIE LIST A	V: Midges (*Culicoides variipennis* spp. *sonorensis* in western U.S., possibly *C. stellifer* in southeastern U.S., also *C. insignis, C. pusillus* in Central and South America) H: White-tailed deer (*O. virginianus*), mule deer (*Odocoileus hemionus*)	Altered geographic distribution of vector species; enhanced vector competence, potential for creating new vector species, increased availability of breeding sites for vectors, increased passive airborne dispersal of vector	CFIA 2001a; Clavijo et al. 2000; Sellers 1992

Table A.8. Cont.

Disease (Agent)	Species at Risk for Clinical Disease	Current Disease Status[1]	Vector (v) and Natural Maintenance Hosts (H)	Effects of Climate Variability and Change	Reference[2]
Equine encephalitis virus (EEE) (Alphavirus)	Horses, humans, some birds (partridge pheasant, emu (*Dromaius novaehollandiae*), ostrich (*Struthio camelus*)	Sporadic in Eastern Canada and U.S. east of the Mississippi River	V Mosquitoes (*Culiseta melanura*, also *Coquillettidia perturbans*, *Aedes vexans*, *Ae. canadensis*, *Ae. sollicitans*, recently *Ae. albopictus* in Florida) H Passerine birds	Altered geographic distribution of vector species; enhanced vector competence, potential for creating new vector species, increased availability of breeding sites for vectors, increased passive airborne dispersal of vector	Carmen et al 1995, Keane and Little 1987, Calisher 1994, Moore et al 1993, Reeves et al. 1994, Sellers 1989, Sellers and Maarouf 1998, 1990, 1993
Epizootic Hemorrhagic Disease (Orbivirus)	Deer, possibly cattle	Western U.S., sporadic incursions into southern BC (Cypress Hills)	V Mosquitoes (*Culicoides variipennis*) H White-tailed deer, mule deer	Altered geographic distribution of vector species; enhanced vector competence, potential for creating new vector species, increased availability of breeding sites for vectors, increased passive airborne dispersal of vector	Pasick et al 2001, Sellers and Maarouf 1991, Sellers 1992
West Nile virus encephalitis (Flavivirus)	Horses, humans, some wild birds, raptors	Introduced into North America in 1999, spread to southern Ontario by 2001; endemic	V Mosquitoes (*Culex* spp., *Aedes* spp.) H Wild birds	Enhanced vector competence, increased availability of breeding sites for vectors, expansion of geographic range	Lopez 2002, Ostlund et al 2000, Epstein 2001, Dohm et al 2002, H. Artsob, pers. comm. March 2002

Table A.9. Potential effects of climate change on parasitic diseases of animals.

Disease (Agent)	Species at Risk for Clinical Disease	Current Disease Status[1]	Natural Maintenance Hosts (H)	Effects of Climate Variability and Change	References[2]
Besnoitiosis (*Besnoitia tarandi*)	Caribou (*Rangifer* spp.), other wild cervids (deer)	Endemic in NWT; rare	H: Carnivores IH: Caribou Mechanical transmission possible by biting flies	Altered range and abundance of definitive and intermediate hosts; possible disease risk to other deer if range overlaps	Leighton 2001; Hoberg et al. 2001; Lankester 2001; Leighton and Gajadhar 2001
Cryptosporidiosis (*Cryptosporidium parvum*)	Young ruminants, humans	Epidemic across Canada and also sporadic; Reportable: in ON	H: Mammals including ruminants, deer, pigs (*Sus domesticus*), foals (*Equus equus*), humans	Waterborne disease associated with contamination by domestic animal waste; flooding may increase risk for waterborne diseases	Jacobs and Medleau 1998
Dioctophymatosis (*Dioctophyma renale*)	Canids, mustelids, rarely humans	Sporadic in ON	H: Mustelids, canids IH: Mudworm (*Lumbriculus variegatus*); Paratenic H: Various species of fish, frogs	Increased rate of development and earlier infectivity of eggs; increased rate of development in mudworm; eggs are intolerant of freezing	Measures 2001; Anderson 2001
Echinococcosis (*Echinococcus granulosus, E. multilocularis*)	Humans	Sporadic in Ontario	H: *E. granulosus*: wolf (*Canis lupus*), coyote (*C. latrans*), dogs (*C. domesticus*); *E. multilocularis*: foxes (*Vulpes* spp.), other canids IH: *E. granulosus*: moose (*Alces alces*), other cervids; *E. Multilocularis*: rodents	Altered range of hosts (e.g., wolf) and intermediate hosts (e.g., moose) may alter prevalence and intensity of infection	Jones 2001
Equine protozoal myelitis (*Sarcocystis neurona*)	Horses	Exposure is common, endemic in ON	H: Opossum (*Didelphis virginiana*) IH: Unknown	Expanded range of opossum in ON	MacKay et al. 2000; Jones and Pybus 2001
Giardiasis (*Giardia duodenalis*, including *G. lamblia*)	Often asymptomatic; host species	Endemic across Canada; Reportable: federally; in ON	H: Dogs, cats (*Felis domesticus*), domestic livestock, wild mammals, humans	Environmental temperatures above 20°C are detrimental to cyst survival; waterborne disease, associated with contamination by domestic animal/human sewage; flooding may increase risk for waterborne diseases	Olson and Buret 2001; Radostits et al. 2000
Hepatic fascioliasis (*Fasciola hepatica*, *Fascioloides magna*)	Sheep (*Ovis aries*), cattle, occasionally wildlife	*F. hepatica*: QC, western AB, Maritimes *Fascioloides magna*: Common in western Canada and ON (Long Point, southern ON)	*F. Hepatica*: H: Sheep, cattle IH: Lymnaeid snails, including *L. columella* *F. magna*: H: elk (*Cervus elaphus*), white-tailed deer (*Odocoileus virginianus*), caribou (usually asymptomatic) IH: Lymnaeid snails	Earlier onset and accelerated life cycles within snail hosts; storms may increase wet habitat available for intermediate snail host	Radostits et al. 2000; Pybus 2001

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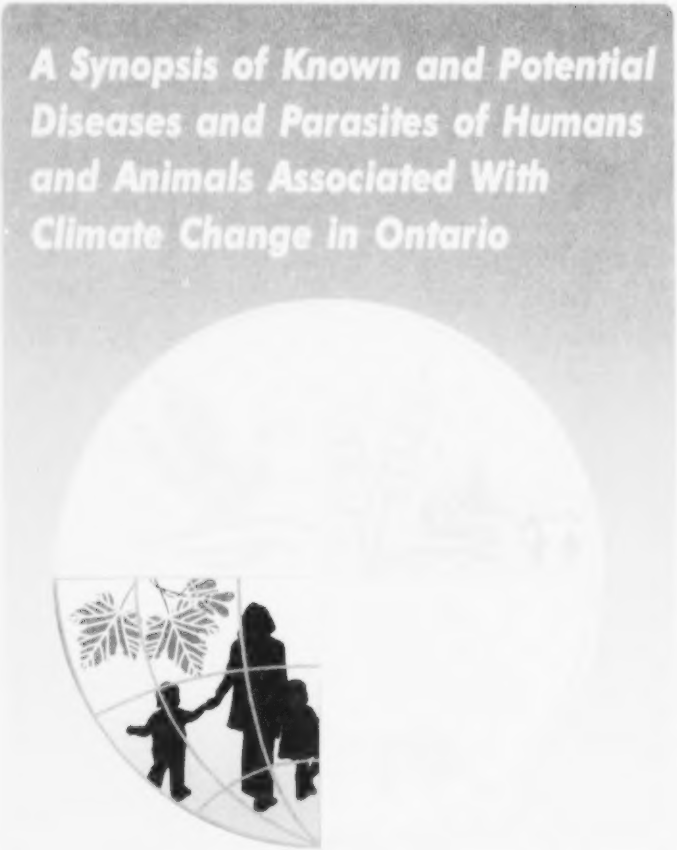
[1] *OIE listing* refers to the Office Internationale des Epizooties classification of diseases. In Canada, all *OIE: list A* diseases are nationally reportable. The *OIE: list B* diseases are of national interest but are not notifiable. The Canadian Food Inspection Agency collects information on OIE: list A and list B diseases.

[2] Full references are listed in Part C of this report.

Table A.9. Cont.

Disease (Agent)	Species at Risk for Clinical Disease	Current Disease Status[1]	Natural Maintenance Hosts (H)	Effects of Climate Variability and Change	References[2]
Larva migrans (*Baylisascaris procyonis*)	Mammals (particularly rodents, rabbits), humans, birds (ratites, domestic poultry)	Sporadic in Canada; highly endemic in eastern Canada	H: raccoon (*Procyon lotor*) IH: Small vertebrates, mammals and birds (wide IH range)	Altered range and abundance of definitive host; increased rate of development of eggs and earlier infectivity of eggs; moisture increases survival of embryonated eggs	Kazacos 2001
Parasitic gastroenteritis (*Trichostrongylus, Ostertagia, Haemonchus*, etc.)	Cattle, sheep	Endemic throughout Canada	H: Ruminants	Enhanced overwintering of eggs larvae, accelerated larval development leading to increased exposure on pastures, improved survival of infective larvae on pasture	Radostits et al. 2000
Parelaphostrongylosis (meningeal worm) (*Parelaphostrongylus tenuis*)	Moose, elk, caribou, sheep, goats (*Capra hircus*), llamas (*Lama glama*)	Endemic with deer	H: White-tailed deer IH: Terrestrial gastropods (snails, slugs)	Altered range and abundance of definitive and intermediate hosts	
Strongylosis, verminous arteritis (Large strongyles) **Gastroenteritis** (Strongyles, cyathostomes)	Horses	Large strongyles: Sporadic across Canada Strongyles: Highly endemic across Canada	H: Horses	Enhanced overwintering of eggs larvae; improved survival of infective larvae on pasture	Hoberg 2001
Taeniasis (*Taenia crassiceps, T. hydatigena*)	*T. crassiceps:* Humans (rarely, intraocular infection), woodchucks (*Marmota monax*) *T. hydatigena:* Subclinical canids, sheep	Sporadic in Canada (*T. hydatigena*); sporadic in ON (*T. Crassiceps*)	H: *T. crassiceps:* canids (fox, wolf, coyote, dog); *T. hydatigena* domestic and wild canids, felids IH: *T. crassiceps:* small mammals (rodents, rabbits, squirrels etc.); *T. hydatigena* domestic livestock, wild cervids	Altered range and abundance of definitive and intermediate hosts; altered range of hosts (e.g., wolf) and intermediate hosts (e.g., moose) may alter prevalence and intensity of infection	Jones and Pybus 2001
Trichinellosis (*Trichinella nativa, T. spiralis*)	*T. nativa:* Usually asymptomatic *T. spiralis:* Humans	Epidemic in the U.S.; OIE LIST B	H: *T. nativa:* polar bears (*Ursus maritimus*), seals, fox, wolf, walrus; *T. spiralis:* Pigs, rodents, humans, black bear (*U. americanus*)	Altered range and abundance of host species; increased survival of larvae in muscle of carrion (intolerant of prolongued freezing)	Dick and Pozio 2001
Verminous pneumonia (*Dictyocaulus viviparous*)	Cattle, farmed elk	Uncommon in ON	H: Cattle	Enhanced overwintering and development of larvae; improved survival of infective larvae on pasture	Radostits et al. 2000
Verminous pneumoniae (*Dictyocaulus filaria, Muellerius capillaris, Protostrongylus rufescens*)	Sheep, goats	Sporadic in ON	IH: Mollusks (*M. capillaris and P. rufescens*)	Enhanced overwintering and development of larvae; improved survival of infective larvae on pasture	Radostits et al. 2000



A Synopsis of Known and Potential Diseases and Parasites of Humans and Animals Associated With Climate Change in Ontario

Part B.

Diseases and Parasites of Humans Associated With Climate Change in Ontario

1. Introduction

Global climate change will have many local impacts in Ontario, including potential implications for the health of Ontarians. Climate change is a sustained departure from normal weather patterns and is widely accepted to be the result of the accumulation of greenhouse gases in the atmosphere. Climate is one of many determinants of health. Various elements of climate interact with the biophysical and social environment in shaping individual and population health. These interactions are complex and dynamic – for example, many infectious diseases cycle from animals through vectors to humans and back into the environment. Climate influences most aspects of these cycles, including the health and reproductive success of the animals, the ability of the vector insects to transmit disease, and human behaviour that results in exposure to infected vectors. Understanding these dynamics and implications of climate change for the future will facilitate the development of adaptive public health and environmental strategies for responding to the health impacts of climate change in Ontario.

Climate change projections for Ontario include warmer temperatures, more frequent extreme weather events, poorer air quality and changes in precipitation patterns, such as heavier rainfall separated by longer dry spells. These changes could lead to increased illness and death from a number of illnesses including heat stress; respiratory disease; some vectorborne diseases, foodborne and waterborne illnesses; and stress-related illnesses due to social and economic disruption. Some Ontarians may be more vulnerable than others to the health impacts of climate change because of immature or malfunctioning immunity (children, the elderly, the sick), occupational exposure (workers in environments without air-conditioning, and farm, bush and other outdoor workers), location (Aboriginal peoples, those in urban, coastal, or remote areas), or social and economic constraints. Yet, climate is only one of many complex, dynamic environmental determinants of health. From agriculture to urbanization, various human modifications of the environment interact with natural ecological systems and affect human health in many ways. In addition, many infectious diseases and parasites of humans also occur in animals or have animal reservoirs. The impacts of climate change on the dynamics of disease in animal populations will alter exposure risks for human populations. Given this complexity, it is difficult to estimate the net impact of climate change on human health. Inferences can be drawn from examining what is already understood about the relationships between climate and disease. For example: More elderly city-dwellers die during heat waves (McNaughton et al. 2002). Foodborne infections generally increase in summer (Isaacs et al. 1998). Fecal contamination of drinking water sources has been shown to increase after heavy precipitation (Auld et al. 2001, Patz et al. 2000, MacKenzie et al. 1994). In Ontario, vectorborne diseases such as Lyme disease and mosquito-borne encephalitis are a rare but exclusively warm-hot weather problem (Leighton 2000b, electronic access).

Lack of clear dose-response relationships between weather parameters and many of the likely health effects and lack of reliable estimates of future regional climates hinder current risk assessments of the regional health impacts of global climate change. A review of the likely regional impacts of global climate change based on a historical perspective (Longstreth 1999), suggests that some regions may be more greatly affected than others, not only because historically they have been more prone to summer weather/climate-related diseases but also because they contain a greater proportion of sensitive subpopulations. Such issues may have important regional implications for Ontario.

Here we present an overview of the potential impacts of climate change on human health in Ontario, with a particular emphasis on the role of weather and climate in the ecology and epidemiology of infectious diseases and parasites. Given the lack of published quantitative analyses on the topic, we have not ventured to assess the relative importance of the projected change in hazard levels from each disease, except where the evidence is substantial enough to justify such speculation. In the concluding section of the combined human and animal disease reports, we present some suggestions for adapting to new or altered health threats wholly or partly attributable to climate change.

2. Direct Effects of Climate on Health

Potential effects of global climate change on human health can be divided into direct and indirect effects, according to whether they occur predominantly through the impacts of climate variables on human biology (direct) or are mediated by climate-induced changes in other biological and biogeochemical systems (indirect). The relatively direct health effects of changes to ambient temperature, frequency of extreme weather, and reduced air quality are addressed in this section.

2.1. Temperature

2.1.1. Thermal Stress From Milder Winters

Global climate change will be accompanied by an increase in the frequency and intensity of heat waves in Ontario, as well as warmer summers and milder winters. Increased humidity may exacerbate the impact of extreme summer heat on human health. Increased frequency and severity of heat waves may lead to an increase in illness and death, particularly among the young, the elderly, and people with pre-existing illness (Patz et al. 2001, McGeehin and Mirabelli 2001). Other vulnerable people are the poor, homeless people, persons with impaired mobility, and those living on the top floor of apartment buildings, in urban areas, and without access to air-conditioning. Excess mortality (deaths beyond those expected for that period in that population) during heat waves is greatest in the elderly and in people with pre-existing illness (Semenza et al. 1996). Much of this excess mortality from heat waves is related to exacerbation of underlying cardiovascular, cerebrovascular, and respiratory disease.

Heat waves also are associated with nonfatal impacts such as heat cramp, heat syncope, heat stroke and heat exhaustion (Patz et al. 2001). Health effects appear to be related to environmental temperatures above those to which the population is accustomed. There does not appear to be a single universal threshold temperature at which rates of heat-related morbidity and mortality rise sharply. Instead, tolerance of excess heat varies regionally according to the population and its preparedness for heat, and according to the local average temperatures and frequency of extreme temperatures. In the United States, models of the weather mortality-relationship indicate that populations in the northeastern and midwestern cities may experience the greatest number of heat-related illnesses and deaths in response to changes in summer temperature and that the most sensitive populations live in regions where extremely high temperatures occur infrequently or irregularly (Kalkstein and Greene 1997). Hajat et al. (2002) had similar findings in London, UK. They found that hot days occurring in the early part of any year might have a larger effect than those occurring later on. Their study also found that heat waves of long duration and of highest temperature have the largest mortality effect.

Heat waves tend to have a much bigger health impact in cities than in surrounding suburban and rural areas (Kilbourne 1997). Urban areas typically experience higher night-time temperatures because of the "heat island" effect, resulting in increased risk for those who lack access to air-conditioning (Smoyer 1998). Air pollution also is typically higher in urban areas, and elevated pollution levels often accompany heat waves (Piver et al. 1999). Urbanized areas in southern Ontario are likely to be at increased risk for heat-related deaths because of the more frequent and severe heat waves anticipated with climate change. Chiotti et al. (2002) employed statistical methods from Kalkstein and Smoyer (1993) to estimate the number of elderly deaths among residents across the Toronto-Niagara region in the future. They found that by 2020, mortality among the elderly could reach from between 171 and 447during an average summer. A recent study of heat-stress-related mortality in five cities in southern Ontario for the years 1980-1996 documented significantly higher mortality among the elderly on heat-stress days (those with an apparent temperature or heat stress index above 32°C) in four of five metropolitan centers (Smoyer et al. 2000). The authors concluded that even without the warming induced by climate change, vulnerability is likely to increase as the population ages and is being intensified by ongoing urban development and sprawl. Other research suggests that Europeans have adjusted successfully to mean summer temperatures ranging from 13.5 °C to 24.1 °C, and can be expected to adjust to global warming predicted for the next half century with little sustained increase in heat-related mortality (Keatinge et al. 2000). This adaptation is dependent on adequate shelter from extremes (both hot and cold) of temperature.

In industrialized countries, shelter from extreme heat generally entails access to air-conditioning, which can considerably reduce the health threat posed by heat waves (Naughton et al. 2002). However, air-conditioning requires a great deal of energy, the creation of which is associated with greenhouse gas emissions, and contributes to urban heat-island effect, feeding the need for additional air-conditioning. While it is true that populations in industrialized nations have become better adapted to the local climate through past technological advances, climate change may pose a significant technological challenge, and additional avenues for adaptation may be required.

2.1.2. Decreased Mortality Resulting From Milder Winters

Extreme cold claims more lives, directly, in Canada than any other extreme weather event (Etkin and Maarouf 1995). Indirect effects of cold, for example, deaths due to weather-related car accidents, are much greater. Prolonged exposure to cold can cause people and animals to suffer hypothermia and frostbite. In general, in Europe and North America, death rates associated with chronic bronchitis, pneumonia, ischemic heart disease and cerebrovascular disease are higher in the winter than in the summer (Langford and Bentham 1995).

The milder winters anticipated for Ontario should be accompanied by a reduction in the number of deaths and injuries that are directly and indirectly attributable to cold. Future increases in heat-related deaths have been predicted to outnumber any decreases in cold-related deaths in the United States by a factor of three (Kalkstein and Greene 1997). However, the weather in Ontario is on average colder than in the United States. The overall balance between changes in summer and winter weather-related deaths is unknown and varies by population and region.

2.2. Extreme Weather Events

Climate change may alter the frequency, timing, intensity and duration of extreme weather events such as storms, tornadoes, floods, and severe drought in Ontario. Over four million Canadians have been affected by natural disasters in recent years, with tragic loss of life and property in the 1996 Saguenay flood, the 1997 Red River flood and the 1998 ice storm in eastern Ontario and western Québec. Growth in urban population and an aging infrastructure may exacerbate the effects of future extreme weather (ICLR 2002, electronic access).

Deaths, injuries, emotional shock and homelessness are the direct health impacts most often associated with natural disasters. Secondary health effects may also occur, mediated by changes in ecologic systems and public health infrastructures, such as infections from unsafe drinking water (Patz et al. 2001). Post-traumatic stress disorders have been reported following natural disasters.

The health impacts of natural disasters include (Noji 1997):

- Physical injury
- Decreased nutritional status, especially in children
- Increases in respiratory and diarrheal diseases resulting from crowded conditions for survivors, often with limited shelter and limited or no access to potable water
- Impacts on mental health, which in some cases may be long-lasting
- Increased risk of water-related diseases as a result of disruption of water supply or sewage systems or direct contamination of wells
- Release and dissemination of dangerous chemicals from storage sites and waste disposal sites into floodwaters

Weather disasters resulting in major disruptions to power service may theoretically lead to outbreaks of foodborne illness resulting from the consumption of spoiled food. Rarely, waterborne outbreaks are reported following climatic disasters in industrialized countries (Lee et al. 1993, CDC 1983) but are considerably more common in developing countries (e.g., Kunii et al. 2002). Inadequate access to potable water may also pose a problem during power outages, since electric well pumps will fail.

Many climate scenarios project increases in the frequency of extreme precipitation events and thus an increased risk of floods (IPCC 2001, electronic access). In Canada, floods are the most frequent natural disaster, as well as the leading cause of death from natural disasters. All rivers are susceptible to flooding, and nearby populations are potentially vulnerable.

Inundations of sites that contain toxic wastes, sewage, animal wastes, or agrochemical products may result in immediate human exposure to contaminated drinking water, contamination of edible fish, and long-term contamination of flooded housing.

Changes in snow accumulation and in the timing of snowmelt are likely to affect the seasonal distribution and characteristics of flood events in some areas (IPCC 2001, electronic access). In southeastern Canadian and northeastern U.S. watersheds, reductions in winter snow and river ice will tend to reduce winter and spring flood risks (Bruce et al. 2000, electronic access). However, rivers in northern areas may begin to experience earlier winter ice break-ups and associated flooding (Bruce et al. 2000, electronic access, IPCC 2001, electronic access).

Milder winter temperatures may decrease heavy snowstorms but could increase freezing rain if average daily temperatures fluctuate about the freezing point (IPCC 2001, electronic access). It is difficult to predict where ice storms will occur and to identify vulnerable populations. However, recent weather trends suggest that parts of Ontario could be at increased risk. The ice storm of January 1998 left 45 people dead and nearly 5 million people without heat or electricity in Ontario, Québec, and New York (Holt et al. 1998, Francis and Hengeveld 1998, Kerry et al. 1999). The storm had a huge impact on human health and on medical services.

Godleski (1997) reports that persons who endure tornadoes often experience a variety of stress responses, including depression, acute and post-traumatic stress disorders, substance abuse, and anxiety. In Canada, during an average year, 80 tornadoes cause two deaths and twenty injuries, plus tens of millions of dollars in property damage. Ontario experiences about 15 tornadoes annually, usually in the southern part of the province. Although most Ontario tornadoes are mild (below F2 on the Fujita severity scale), they may occasionally reach F4, causing major property damage (Environment Canada 2002, electronic access). Tornadoes, because of their sudden nature, are weather events most likely to result in unexpected disaster. Existing climate models do not allow the projection of the effects of climate change on the frequency and intensity of tornadoes in Ontario or elsewhere (Greenough et al. 2001).

Summer convective storms in Ontario are already common and may increase with climate change. Although direct injury and trauma from lightning strikes are rare, indirect injuries or deaths may occur from fires due to lightning and from storm-related damage to structure and trees. People with allergies to grass pollen are at risk of thunderstorm-related asthma (Venables et al. 1994, Celenza et al. 1996). Thunderstorm-associated asthma epidemics have placed considerable demands on the health system. In 1994, several London, UK health departments ran out of drugs, equipment, and doctors due to an epidemic of thunderstorm-associated asthma (Davidson et al. 1996). It is unclear if this situation has ever arisen in Ontario or if it could become a common problem in future years.

2.3. Effects of Reduced Air Quality

Exposure to air pollutants has serious public health consequences and is a significant problem in southern Ontario. The city of Windsor has the poorest air quality in the country. More than any other region or province, southern Ontario is subjected to poor air quality periodically throughout the warmer months (OMA 2001, electronic access). Mean annual ground level ozone levels have increased by 0.5% annually since 1979 (OMA 2001, electronic access) though the relatively cooler temperatures in 2000 resulted in lower levels of ozone than predicted (OMA 2001, electronic access).

In addition to ground-level ozone, air pollutants include other toxicants, acid precipitation, and particulates. As the effects of climate change on these pollutants are unknown, it is difficult to determine the overall effects of climate variability and change on respiratory health (Patz et al. 2001). However, some inferences may be made based on what knowledge is available.

Weather conditions influence air pollution via pollutant transport and/or formation. Weather conditions also can influence biogenic (e.g., pollen) and human-caused (e.g., fossil fuel) air pollutant emissions (IPCC 2001). For example, large high-pressure systems often create an inversion of the normal temperature profile, trapping pollutants in the shallow boundary layer at the Earth's surface; higher temperatures favour smog formation. Precise projections of the impacts of climate change on average local air pollution concentrations are not yet possible. However, any increase in heat and anti-cyclonic conditions in summer would tend to increase air pollution concentrations in cities (IPCC 2001, electronic access).

Ground-level ozone and suspended particulates are significant contributors to smog and poor air quality and have a range of respiratory and cardiovascular health effects (Duncan et al. 1998, Stieb et al. 1996). Climate change is likely to worsen the amount of suspended particulates (Maarouf and Smith 1997). Climate change may also increase the concentration of ground-level ozone, but the magnitude of the effect is uncertain (Patz et al. 2000). Formation and destruction of ozone is accelerated by increases in temperature and in the intensity of ultraviolet radiation. Existing air quality models indicate that decreases in stratospheric ozone and elevated temperature will increase ground-level ozone concentrations (IPCC 2001). An increase in hot days could increase emissions of volatile organic compounds from all sources (Sillman and Samson 1995). Taken together, these findings suggest that poor air quality will continue to be a problem in Ontario during heat waves and that this problem may worsen with climate change.

The increased frequency of convective storms projected under climate change scenarios for Ontario could affect the potential for airborne pathogen dispersal. Table B.1 lists some of the pathogenic bacteria, fungi and viruses that are transmitted through airborne transport and occur in Ontario. Most airborne bacterial and viral diseases of concern in Ontario are transmitted by droplet aerosol (coughing, sneezing, etc). Impacts of climate change on the transmission of such diseases might be indirect: For example, hotter summers could result in more time spent indoors, thus increasing exposure to re-circulated air and providing more opportunities for respiratory virus transmission. Changes in temperature and precipitation may affect the infectivity of pathogenic fungi in the environment (Stephen et al. 2002), although the implications for fungal diseases in Ontario are not clear. Although Hantavirus may be transmitted by airborne dust contaminated by mouse droppings (for example, by inhaling while sweeping contaminated areas), the disease is more appropriately discussed in the section of this report that focuses on other rodent-borne diseases.

Arthropod vectors can also be dispersed by wind (Sellers 1989, Sellers and Maarouf 1990) and are discussed in the vectorborne disease section of this report. Many fungi and viruses such as the Hantavirus are typically transmitted in association with airborne dust. While most

Table B.1. *Some human airborne pathogens (from Griffin et al. 2001).*

Agents	Disease
Bacterial Agents	
Bordetella pertussis	Whooping cough
Corynebacterium diphtheriae	Diphtheria
Legionella pneumophila	Pontiac fever, Legionnaire's disease
Haemophilus influenzae, Neisseria meningitides, Streptococcus pneumoniae	Bacterial flu, bacterial meningitis
Mycobacterium tuberculosis	Tuberculosis
Fungal Agents	
Aspergillus spp.	Aspergillosis
Coccidioides immitis	Coccidiomycosis
Histoplasma capsulatum	Histoplasmosis
Blastomyces dermatitidis	Blastomycosis
Viral Agents	
Rhinoviruses	The common cold
Influenza viruses	Viral flu
Herpes virus-3	Chicken pox
Hantavirus	Hantavirus pulmonary syndrome

airborne pathogens may be transmitted over relatively short distances, recent evidence indicates that processes that contribute to desertification in remote areas may also contribute to long-range atmospheric transport of dust and associated airborne pathogens (Griffin et al. 2001). This mechanism could theoretically be responsible for the future introduction of new diseases to Ontario.

Based on an estimated prevalence of allergic rhinitis of 10-20%, airborne allergens affect the health of hundreds of thousands of Ontarians from spring through late summer (AAISO 2002, electronic access). Daily, seasonal, and inter-annual variation in the abundance of many aeroallergens, particularly pollen, is associated with meteorological factors (Emberlin 1994, Celenza et al. 1996). While pollen abundance is more strongly associated with land-use change and farming practices than with weather (Emberlin 1994), climate change may affect the length of the allergy season or alter the geographic distribution of plant species (Patz et al. 2001). In addition, higher ambient levels of CO_2 may increase pollen production. Experimental research has shown that a doubling in CO_2 levels, from about 300 to 600 ppm, induces an approximately four-fold increase in the production of ragweed pollen (Ziska and Caulfield 2000a,b). With climate change, allergic rhinitis could pose a larger health burden in Ontario.

2.4. Effects of Ultraviolet Light and Stratospheric Ozone Depletion

Stratospheric ozone destruction is a separate process from greenhouse gas accumulation in the lower atmosphere. In the 1980's, depletion of stratospheric ozone was linked to chlorinated hydrocarbon emissions. Reducing the use of these gasses worldwide has decreased the rate of stratospheric ozone depletion. Although climate change will not directly contribute to stratospheric ozone depletion, several of the anthropogenic greenhouse gases (e.g., chlorofluorocarbons and nitrous oxide (N_2O)) are also ozone-depleting gases. In addition, tropospheric warming (from greenhouse gas accumulation) induces stratospheric cooling, which exacerbates ozone destruction (Shindell et al. 1998, Kirk-Davidoff et al. 1999). Cloud cover patterns are expected to be altered by climate change, which in some places may mitigate damaging ultraviolet radiation (UVR) by providing cover and in other places may result in more UVR reaching the planet's surface because cloud cover is reduced. The 1-2% increased cloud cover projected for Ontario (Smith et al. 1998) seems unlikely to have a significant impact on human exposure to UVR.

Stratospheric ozone shields the Earth's surface from incoming solar UVR that can harm human health. Long-term cyclical summertime decreases in stratospheric ozone over New Zealand have been associated with significant increases in ground-level UVR, particularly in the DNA-damaging waveband (McKenzie et al. 1999). In a warmer climate, patterns of personal exposure to solar radiation (e.g., sunbathing in temperate climates) also are likely to change. Many epidemiological studies have implicated solar radiation as a cause of skin cancer (melanoma and other types) in fair-skinned humans (IARC 1992, WHO 1994). The most recent assessment by UNEP (1998) projects significant increases in skin cancer incidence worldwide because of stratospheric ozone depletion.

The formation of lenticular cataracts may also be exacerbated by increased UVR. However, its role in cataract formation is complex and only some cataract subtypes appear to be associated with exposure to UVR.

In humans and experimental animals, UVR can cause local and whole-body immune suppression (UNEP 1998); ambient doses of UVR reduce cellular immunity (Garssen et al. 1998). Concern exists that such immune suppression could increase susceptibility to infection and alter the distribution of infectious diseases. Nevertheless, no direct evidence exists for such effects in humans, and uncertainties remain about the underlying biological processes.

3. Indirect Effects of Climate Change on Health

The indirect health effects of climate change include a wide range of infectious diseases, parasites, and nutritional health issues. The impacts of climate change on these illnesses will be mediated by changes in the environment, carrier animal and vector populations, and human behaviour, rather than the more direct impacts discussed previously. These changes in disease ecology are more complex and thus more difficult to project than direct health impacts of climate variability or change.

3.1. Vectorborne Diseases

Vectorborne diseases are transmitted to humans by some intermediate host, often an insect. Disease vectors are generally sensitive to climate, because their temperature, humidity, and habitat needs are usually highly specific. Because of Ontario's temperate climate and good public health and sanitary infrastructure, vectorborne diseases are generally uncommon here. However, some vectorborne diseases have recently become established in Ontario, such as Lyme disease and West Nile virus (Barker 2000, HC-NACI 2000, electronic access). Climate change will likely affect Ontario's vulnerability to these and other vectorborne diseases.

Temperature and humidity greatly influence vector biology and ecology. Many vectors prefer particular microclimates (the immediate environment occupied by an individual insect). Climate change can have a range of potential direct and indirect effects on vector biology, on interactions between vectors and disease agents, and on habitat and microclimate. Since these effects interact and are highly complex, the outcomes are difficult to project, but they are likely to be influenced by sustained change in climate. A range of climatic effects on vector and disease ecology is summarized in Appendix 2.

Projected temperature increases will probably lengthen the theoretical transmission season in Ontario for all vectorborne diseases. Actual risk of the disease depends on factors such as mosquito control programs, insecticide and drug resistance, human behaviour, land use and access to health care (Patz et al. 2000), in addition to transmission season length. For effective transmission, conditions must favour increased human-vector contact, increased survival and density of hosts or reservoirs, and co-maturation of vector and disease agent. Sustained changes in temperature, humidity, or precipitation could alter the distribution of existing vectorborne diseases in Ontario (such as Lyme disease) and facilitate the establishment of new diseases. Furthermore, changing wind patterns and extreme weather events such as convective storms may alter airborne vector dispersal patterns. Mosquitoes infected with arboviral encephalitides (western and eastern equine, St. Louis) could be blown into Ontario from the United States and under the right climatic conditions could cause disease outbreaks. Thus, under the projected climate change scenarios put forward by the IPCC, Ontarians could face an increased risk from certain vectorborne diseases, including arboviral encephalitis, rickettsial diseases, and Lyme borreliosis. Vectorborne diseases are summarized in Table A.2.

3.1.1. Arboviral Encephalitis

Dozens of infectious agents may cause encephalitis in humans. Enterovirus infection is the most common cause of encephalitis. Less common, but still significant, is infection by one of several arthropod-borne viruses, or arboviruses. They are members of three virus families, and include Togaviridae (genus Alphavirus), Flaviviridae, and Bunyaviridae (Griffin 2000). Viruses that have posed a threat to Ontarians in the past or that might become a threat in the future include the alphaviruses western and eastern equine encephalitis virus (WEE, EEE), the flaviviruses St. Louis Encephalitis (SLE), West Nile (WNV), and Powassan encephalitis (POW), and the bunyaviruses classified as California viruses, including LaCrosse (LAC) virus and snowshoe hare (SSH) virus (CDC-DVID 2002a, electronic access).

Symptoms of viral encephalitis infection may range from mild flu-like symptoms to severe encephalitis. Encephalitis is a general term used to describe inflammation of the brain; encephalomyelitis refers to inflammation of the brain and spinal cord. Encephalitis is always a serious illness, often resulting in hospitalization of the victims, and can result in death. There is no specific treatment for viral encephalitis and vaccines are not widely available for use in humans except for Japanese encephalitis (Griffin 2000). Historically, arboviral encephalitis has been a rare, late summer occurrence in Ontario (Artsob 2002, pers. comm.).

Arboviral encephalitides are maintained in complex life cycles involving nonhuman primary hosts (usually birds, rodents) and primary arthropod vectors (a mosquito or a tick, for example). The virus is maintained in the wild vertebrate host population, often remaining undetected until humans are affected. This detection may occur when people move into an area that is a natural focus of disease in wildlife or when the virus escapes this focus via a secondary vector or vertebrate host due to some ecologic change. Although humans can become very ill, a significant viremia may fail to occur, blocking transmission from humans to others. Thus humans rarely serve as a source of infection to others, except rarely by transfusion.

Forecasts under conditions of global warming suggest a potential for increased arboviral encephalitis throughout the United States (Longstreth and Wiseman 1989, Shope 1990). The role of climate in arbovirus transmission is extremely complex. However, some broad trends may be anticipated. Increased temperatures and longer summers will likely extend the winter survival of mosquitoes in southern Ontario, extend the summer transmission season, and allow the ranges of some disease vector species now absent from Ontario to expand north. Together with accelerated intrinsic incubation periods for the viruses within the vectors, these changes in vector ecology might play a role in the re-emergence of some arboviruses in Ontario and in the potential introduction and spread of new arboviruses (for example, WNV). Drier conditions in summer may reduce the local mosquito burden; however, extreme precipitation events will create intermittent puddles and other mosquito habitat.

The main vectors of the arboviruses that circulate in avian hosts (WNV, WEE, EEE, SLE) are mosquitoes that feed principally on birds. Effective transmission of the viruses to humans or horses requires infection of bridging vectors which feed on birds and mammals, allowing the virus to jump from the reservoir population to the susceptible mammal population. Population and disease dynamics and response to climate change (longevity, infectivity, etc.) may be different for endemic and potential bridging vectors, compounding the complexity of the situation. Factors limiting establishment of an endemic focus of these viruses in Ontario are unknown but may relate to temperature or other constraints on vectors and on virus-vector interactions.

a) St. Louis Encephalitis

St. Louis encephalitis (SLE) virus is a flavivirus related to the Japanese encephalitis virus. The virus cycles between wild birds and mosquitoes (*Culex* spp.) that prefer birds, and is occasionally transmitted to humans by infected mosquitoes. The incubation period is between 5 and 21 days. Human case fatality rates are in the range of 3 to 30% (Tsai 2000). The virus is highly prevalent in the United States; the California group viruses are more common, and West Nile virus is rapidly becoming the most common arboviral encephalitis in the United States. There are usually under 50 cases reported per year in the United States, with sporadic epidemics causing several hundred cases, primarily in the midwestern and southeastern states. The most recent American outbreak was in 1990 (CDC-DVID 2002g, electronic access).

The last major Canadian outbreak of St. Louis encephalitis occurred in 1975 in southern Ontario (Windsor-Sarnia-Chatham area, the Niagara region and the city of Toronto), apparently an extension of the outbreak that occurred in the midwestern United States that summer (Spence et al. 1977).

Some climatic changes projected for Ontario and elsewhere may alter the distribution of SLE in North America. It has been projected that SLE would cease to be endemic in the southwestern United States due to excessively high heat and would move northward (Reeves et al. 1994). Local conditions that promote mosquito survival and multiplication include warm temperatures, absence of excess heat, adequate humidity, and standing warm water for larval survival. Milder winters and longer summers will extend the mosquito season and SLE transmission season in Ontario. Warmer summer temperatures will enhance mosquito reproduction and virus replication; however, extreme heat will kill mosquitoes.

b) West Nile Virus

West Nile virus (WNV) is also a flavivirus of the Japanese encephalitis group. WNV was first isolated in the West Nile province of Uganda in 1937 (CDC-DVID 2002i, electronic access). Until recently, the disease had been reported only in Europe, the Middle East, western Asia, and Africa. The virus was first found in North America in association with an outbreak of encephalitis (at first suspected to be SLE) in New York City and the surrounding area in late August and September 1999 (Lopez 2002). In 2001, the virus was detected in dead birds in southern Ontario, and in 2002, it caused illness and death in people in Ontario and elsewhere in Canada (Figure B.0). A cycle of transmission between wild birds and native mosquitoes has evidently become established in North America, with migratory birds likely playing an important role in the natural transmission cycles and spread (HC-BID 2001, electronic access). Although it is

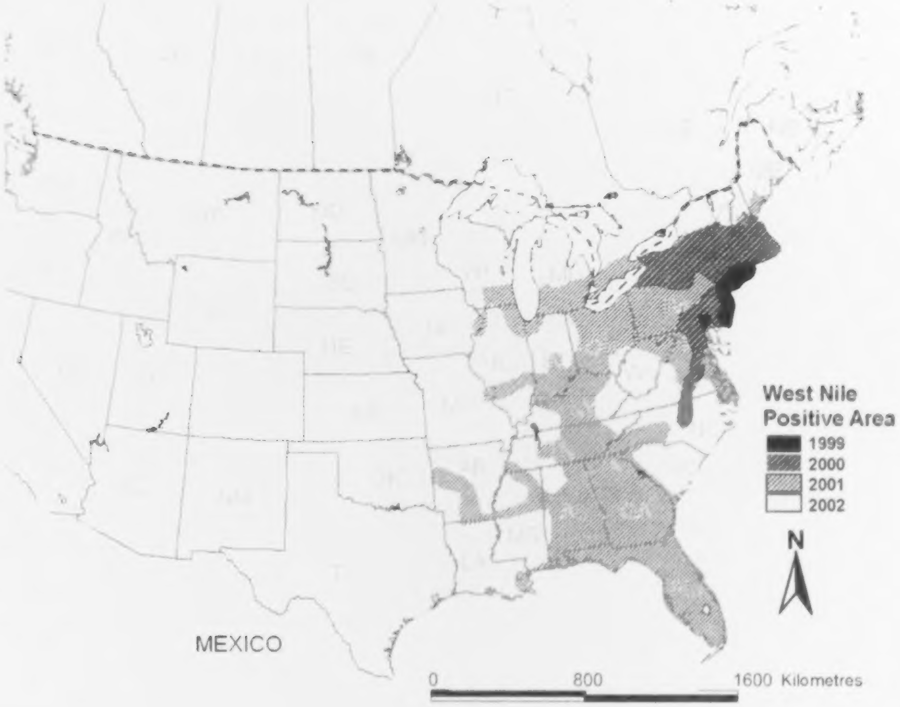


Figure B.0. *Spread of West Nile virus in North America (Rob Meyers, personal communication).*

not known when and how West Nile virus was introduced into North America, international travel of infected persons to New York or transport by imported infected birds may have played a role.

Like the SLE virus, WNV is transmitted principally by *Culex* mosquito species but can also be transmitted by other mosquito species such as *Aedes*, *Anopheles*, and others (OMHLTC 2002a, electronic access). WNV in humans usually produces either asymptomatic infection or mild fever, but occasionally (1 in 150 infections) can cause severe and fatal encephalitis (Petersen and Marfin, 2002). The clinical severity of infection may be worsening, as evidenced by large numbers of cases and fatalities in the United States during 2002. The elderly are most susceptible to neurological illness and to worse outcomes when ill with WNV. The incubation period for WNV acquired by mosquito is five to fourteen days (Petersen and Marfin, 2002). WNV transmission has occurred through organ transplantation, blood transfusion (CDC 2002c), and transplacental transmission (CDC 2002d). Transmission through breast milk has been suggested as well (CDC 2002e).

The virus may be able to survive an Ontario winter, as suggested by the detection of an infected crow early in the transmission season (found in Peel, May 19, 2002) following the exceptionally mild winter of 2001-02. The first human case of WNV in Canada was confirmed on September 6, 2002 in Halton Region, Ontario. Many other human cases have since been reported, in Ontario and elsewhere in Canada. In total, there were 119 confirmed and 182 probable cases of WNV in Ontario in 2002. There has been one confimed death, and several more deaths of confirmed and probable cases are under investigation (Government of Ontario 2002, electronic access). Human cases of WNV were reported in Ontario, Quebec, Manitoba, and Alberta in 2002. The Alberta case was linked to travel to the southern United States (HC-CCHO 2002, electronic access). Infected birds have been detected in all these provinces prior to outbreaks of illness in humans. Infected birds were also found in Saskatchewan in August 2002 and in Nova Scotia in September 2002. During 2002, there were 3,955 cases and 252 deaths reported in 40 of the United States (CDC-DVID 2002i, electronic access).

Climatic conditions, including a mild winter coupled with prolonged drought and heat waves, may have played a role in the initial outbreak of WNV (Epstein 2001), favouring breeding of the principal vector, *Culex pipiens*, in urban environments and accelerating the development of virus within the mosquito. Recent studies have confirmed that elevated temperatures increase the vector competence of *C. pipiens* for WNV (Dohm et al. 2002), possibly contributing to the explosive transmission of this virus in New York during the summer of 1999, a year of near-record-high temperatures. Persistence of the virus and its spread to other states and across Canada suggest that WNV is now endemic in North America and that its geographic range probably will continue to expand. In North America, the virus is affecting many new species, including raptors that have experienced significant mortality. The virus may not behave ecologically as it has in Europe, Asia Minor, and Africa.

Interestingly, the activity of SLE virus in Ontario in 1975 and that of WNV in 2001 covered similar geographic regions in Ontario (Artsob 2002, personal communication). Both viruses use the widespread *C. pipiens* as their principal endemic vector, and climate factors likely influence the distribution of the virus over the range of vectors. Since WNV is new to North America, has spread so rapidly, and has affected so many new avian and mammalian species, it may serve as an excellent subject of study in anticipation of future infectious disease introductions, including those attributable to climate change. Experience from the 2002 season suggests that the virus has become established in Ontario. Climate change projected for Ontario may contribute to the further establishment and spread of WNV and provide climatic conditions appropriate for other mosquito-borne viruses. The advent of WNV in Canada provides us with an opportunity to gain a better understanding of how other zoonotic vectorborne diseases might behave under conditions of climate change, in particular diseases with an avian reservoir. These include eastern equine encephalitis (EEE) virus and western equine encephalitis (WEE) virus (discussed below), as well as SLE.

c) Powassan Encephalitis, Deer-Tick Virus (Tickborne Encephalitis)

First described in Ontario, Powassan (POW) virus is a flavivirus and the first well documented tickborne encephalitis occurring in North America. Recently, a related flavivirus, provisionally named deer-tick Virus (DTV), has been isolated from black-legged ticks (*Ixodes scapularis*) from New England and Wisconsin (Telford et al. 1997, Ebel et al. 1999). These belong to a group of viruses collectively known in the circumpolar nations to cause tick-borne encephalitis (Tsai 2000). POW virus is transmitted by several species of tick, principally *Ixodes cookei* but also *Ixodes marxi*, the wood tick (*Dermacentor andersonii*), and recently the black-legged tick, the vector for Lyme disease (Costero and Grayson 1996). Transmission of DTV is exclusively by the black-legged tick (Telford et al. 1997).

POW virus is maintained in several animal host populations, including groundhog (*Marmota monax*), chipmunks (*Eutamius minimus*), squirrels, snowshoe hare (*Lepus americanus*), and skunks (Leighton 2000b, electronic access). Humans become infected by tick bites, with an incubation period of 7-14 days. Although the virus is widespread in the reservoir population, it only very rarely causes acute viral encephalitis in humans, with one person becoming ill for every 250 infected. The illness is biphasic, with neurological symptoms appearing in 5 - 30% of patients, but being quite severe in almost 50% of these patients. The case fatality rate varies between 0.3% and 60% (the highest case fatality rate among arboviruses). Infection, whether unapparent or overt, leads to immunity (Tsai 2000). The clinical characteristics of DTV are not well described, but it is suspected that DTV infection usually results in mild or sub-clinical illness (Ebel et al. 1999).

POW virus was first isolated from the brain of a five-year-old child from Powassan, Ontario, who died in 1958 (McLean and Donohue 1959). In Canada, 12 cases have occurred between 1958 and 1999, mostly in young children (Leighton 2000a, electronic access). The disease was fatal in at least four cases. A 1959-1961 study in northern Ontario found the prevalence of POW infection in wild mammals to be 0.5% to 3.3% (CDC-DVID 2002a, electronic access). In 1985, a serological survey of several wild mammal species in Ontario including coyote (*Canis latrans*), red fox (*Vulpes vulpes*), raccoon (*Procyon lotor*) and skunks revealed a moderate to high frequency of antibodies to Powassan virus and the snowshoe hare virus (a California virus). These animals support the endemic presence of these viruses in Ontario

(Artsob et al. 1986). Four new human cases of POW encephalitis were diagnosed in Maine and Vermont over the period of 1999 to 2001. Diagnosis of these cases was likely because of increased arbovirus surveillance in response to the WNV outbreak and may not represent a new cluster of disease (Courtney et al. 2001).

Despite being widespread in reservoir host populations, POW is rarely detected as a cause of disease in humans. This may be because people are not often exposed to infected ticks in their natural environment; infected ticks and nymphs rarely select human hosts; the virus is poorly infective to humans; and/or many cases escape diagnosis. Longer and warmer summers may expand tick habitat and host range and reduce winter mortality of tick hosts. These changes, along with more human recreational activity during longer summers, could increase Ontarians' risk of contracting tick-borne encephalitis and other diseases.

d) Western and Eastern Equine Encephalitis

Eastern equine encephalitis (EEE) virus and western equine encephalitis (WEE) virus are alphaviruses closely related to Venezuelan equine encephalitis (VEE) virus. Like the flaviviruses, they are arthropod-borne. This group of viruses is capable of causing severe illness in horses (*Equus equus*), as well as humans. EEE virus cycles between wild birds and *Culiseta melanura* mosquitoes. WEE also circulates in wild birds and is transmitted by *Culex tarsalis*. Infection with either encephalitis virus does not generally cause illness in birds. The vectors are associated with irrigated agriculture and stream drainages (CDC-DVID 2002a, electronic access). The disease is occasionally transmitted to humans (and other mammals) by other mosquito species that feed on birds and humans (Markoff 2000). It is not known how the virus survives over winter, but it may be re-introduced every spring with migratory birds.

The incubation period for the disease in humans is between four and 10 days (Markoff 2000). In humans, EEE virus causes severe disease more often than SLE or WNV does, with human case fatality rates in the range of 33% and permanent brain damage occurring frequently in survivors. WEE does not generally cause severe disease in people. Very young children are affected more severely than adults, with permanent sequelae seen in 5 to 30% of affected children. The mortality rate approaches 3% (up to 15% of clinical cases of WEE) (CDC-DVID 2002a, electronic access). Human EEE and WEE cases are usually seen in early June or July (Leighton 2002a, electronic access).

Horses are highly susceptible to WEE and EEE. Equine epizootics have been reported historically for EEE in Ontario and Québec and for WEE in areas of Manitoba adjacent to Ontario (Leighton 2000b, electronic access). Because horses are outdoors and attract mosquitoes, they are at high risk of infection when the virus is present in mosquitoes. Sick horses are not a source of infection for people, although disease in horses may serve as a sentinel for risk to people since human cases are usually preceded by larger outbreaks in non-vaccinated horses. Routine annual vaccination of horses is recommended (AAEP 2002, electronic access).

Cold weather in the fall ends the mosquito-bird infection cycles for WEE and EEE, as mosquitoes die or become dormant. Sporadic outbreaks of WEE have been recognized in Canada in each decade since 1930. An epidemic occurred in the Prairie Provinces in 1941 in which 1094 human cases were recorded, which is most of the total of cases (1500 or so) recorded in Canada up to 1990.

The distribution and prevalence of WEE and EEE in Ontario are not well defined, but the diseases are rarely reported. No antibodies to WEE or EEE were detected in a serological survey of 725 sera from several wild mammal species in Ontario (Artsob et al. 1986). The current regions of enzootic EEE virus are the American Great Lakes region and the Atlantic and Gulf coasts (Calisher 1994, Moore et al. 1993). WEE virus has been reported from Ontario in the Lake Superior area, but it is found more frequently further west. Disease in humans and horses seems centred on Saskatchewan and Manitoba, where one vector, *Culex tarsalis*, is widely distributed in agricultural areas. Other mosquito species capable of transmitting this virus include *Aedes, Anopheles, Coquillettidia, Culex* and *Culesita*. Because the virus replicates effectively in birds, birds become amplifiers of the virus and important sources of infection for mosquitoes. House sparrows (*Passer domesticus*) and house finches (*Carpodacus mexicanus*), which are expanding their range in Ontario, are important amplifying hosts in western Canada. This virus also infects snowshoe hares, garter snakes (*Thamnophis* spp.) and leopard frogs (*Rana pipiens*). These birds and other animals are widespread in Ontario.

Elsewhere, backward trajectory analysis of wind patterns has provided evidence for windborne transport of mosquitoes infected with alphaviruses from more southerly locations (Sellers 1989, Sellers and Maarouf 1990). Analysis of meteorological patterns has also shown that outbreaks of EEE have been associated with warm wet summers along the east coast of the United States (Freier 1993).

Modelled correlations between wind trajectories and the locations of epidemics of WEE in Manitoba, Minnesota and North Dakota support the theory that aerial transfer of infected *Culex tarsalis* mosquitoes might occur from Texas, via Oklahoma, Kansas and Nebraska (Sellers and Maarouf 1993). In the models, distances of 1250 to 1350 km were theoretically traversed in 18 to 24 hours at heights up to 1.5 km and temperatures greater than or equal to 13°C, and landing was modelled to occur where the warm southerly winds met cold fronts associated with rain. Similarly, trajectory analysis models of winds supported the distribution of EEE from infected mosquitoes (likely *Culiseta melanura*) in the southeastern United States, along the Gulf and Atlantic coasts and up the Mississippi Valley, to upstate New York and Michigan (Sellers and Maarouf 1990). This range acts as a reservoir for potential spread into Ontario.

As with other arboviruses, the occurrence of WEE and EEE may be influenced by projected climate change for Ontario. The largest outbreak of vectorborne disease in humans in the United States following a natural disaster was an outbreak of WEE following the Red River flood of 1975 (Nasci and Moore 1998). It is not clear why WEE and EEE are not more widespread in Ontario given current climate conditions, adding to the difficulty in projecting the potential impact of climate change on their future ranges.

c) Vectorborne Bunyaviruses (California Serogroup)

This group of zoonotic bunyaviruses includes LaCrosse virus (LAC), snowshoe hare virus (SSH), Jamestown Canyon virus (JC), and others. LAC encephalitis is the most common central nervous system disease of children in the United States. The California serogroup encephalitides were the most commonly reported arboviral infections in the United States (HC-PPHB 1996, electronic access) prior to the emergence of WNV. LAC is distributed across the north-central and northeastern United States and is transmitted by the forest mosquito *Aedes triseriatus*, with a reservoir in number of rodents and foxes (Glass et al. 2000). The disease is not commonly seen in Ontario.

Infection of people with SSH virus has been documented in Ontario and elsewhere in Canada. Non-fatal clinical illness has also been reported in Ontario. In 1985, there had been 15 cases of SSH and one case of JC in Ontario (Keane et al. 1988). Other surveys in Canada have found that 0.5% - 32% of people possess antibodies to California group viruses; in most of these people the virus would have been SSH. The virus does not appear to cause disease in wildlife or livestock. The virus is present in snowshoe hare and many other small mammals. There are many competent vectors for the virus, including mosquitoes of the species of *Aedes*, *Culiseta* and *Culex*. Species of *Aedes* have had the highest rates of infection (Leighton 2002a, electronic access).

Although climate change may alter the transmission dynamics and ecology of these diseases, their absence from or rarity in Ontario and/or their mild symptoms (e.g., SSH) may not alter their minor public health significance.

3.1.2. Lyme Disease

Lyme disease is a tickborne zoonotic bacterial infection caused by *Borrelia burgdorferi sensu lato*. *B. burgdorferi sensu lato* refers to 10 bacterial genospecies, of which three are pathogenic to people: *B. burgdorferi sensu stricto*, *B. garinii* and *B. afzelii* (Baranton et al. 1998). Populations of infected *Ixodes* ticks are endemic in parts of southern Ontario and coastal and central British Columbia. Sporadic occurrences of infected ticks have also been reported in Saskatchewan, Manitoba, Quebec and Nova Scotia (Lindsay et al.1999, Morshed et al. 2000, Lindsay et al. 1995).

Reproducing populations of competent vectors for *B. burgdorferi* are confirmed to be established in only two regions of southern Canada: (1) *Ixodes pacificus* is present in regions of the Fraser delta, the Gulf Islands, and Vancouver Island; and (2) the black-legged tick is established along the north shore of Lake Erie (Long Point, Point Pelee, Rondeau Provincial Park (Lindsay, pers. comm.). Small mammals, particularly white-footed mice (*Peromyscus leucopus*) in Ontario are the main hosts for immature tick stages and serve as reservoirs for *B. burgdorferi*, while white-tailed deer (*Odocoileus virginianus*) are the most important hosts for adult ticks in Ontario (Lane et al. 1991, Lindsay et al. 1995). *B. burgdorferi* has been recovered from other arthropod vectors (Lindsay et al. 1998) though trans-stadial competence has not been demonstrated.

Approximately half of the 15 to 40 cases of Lyme disease reported annually in Ontario are acquired in the province (OMHLTC 2002b, electronic access). Lyme disease is a provincially reportable disease in Ontario. Despite the relatively small number of reported cases, the disease garners considerable attention, in part because it is much more common in the neighbouring United States. Lyme disease is the most common vectorborne disease in the United States with over 16,000 cases annually (CDC-DVID 2002e, electronic access).

Barker and Lindsay (2000) reported that the incidence of Lyme borreliosis in Ontario (1988-1998), including cases acquired out of province, was 0.2 per 100,000 population, ranging from 0.4 per 100,000 population in the northwest, to 0.2 per 100,000 population around Long Point, to 0.1 in the rest of the province. A similar mean annual incidence is reported in states where there are few established populations of *I. scapularis* including Michigan, Illinois, and Ohio. Incidence can be much lower than the annual mean in areas where vector populations are widespread and moderately high such as Minnesota, Wisconsin and New York (5 to 25.51 in 1998), or where they are very high such as Connecticut and Massachusetts (105 to 1200 in 1998) (see Barker and Lindsay 2000 for complete data). In the United States, the disease is most prevalent in the Atlantic and upper north-central regions, and in northwestern California. The number of annually reported cases of Lyme disease in the United States has increased about 25-fold since national surveillance began in 1982, and it is now the most significant arthropod-borne disease in the United States, Canada and Europe, in terms of human cases. (This relative importance is changing as WNV becomes established in North America.)

According to *Mandell, Douglas, and Bennett's Principles and Practice of Infectious Diseases*, the classical human clinical syndrome of Lyme disease includes an initial target-shaped rash around the tick bite (*erythema migrans*), followed by a systemic illness with a variety of symptoms, typically including arthritis. The incubation period of the disease varies. If the classic rash is missed or does not appear, other symptoms may not develop until several months or longer after the tick bite. Diagnosis is by recognition of classical skin lesion and by serological testing. The disease may cause chronic arthritis, nervous systems disorders, dermatitis, and debilitation (Steere 2000). Lyme disease can be treated with antibiotics at all stages. In rare cases, antibiotics may not be effective. A vaccine that offers some protection is licensed for use in Canada (HC-NACI 2000, electronic access).

Pet dogs (*Canis domesticus*) may be a source of human exposure to infected ticks. Although dogs are susceptible to Lyme disease, canine infection is usually asymptomatic. Serological monitoring of the disease in dogs may prove a useful sentinel for increased human risk (Banerjee et al. 1996).

Lyme disease is endemic and relatively common in northeastern United States (CDC-DVID, 2002e, electronic access). The range of the black-legged tick in the United States is expanding rapidly. It is possible that a similar but much smaller expansion in the range of Lyme disease may be occurring in Ontario, with implications for exposure to humans. Established populations of infected ticks are now detected at Long Point, Point Pelee National Park and Rondeau National Park (Barker and Lindsay 2000, Banerjee et al. 2000). There is evidence that birds may drop infected ticks throughout the province during spring migration (Klich et al. 1996), thus contributing to the sporadic occurrence of the disease in Ontario.

The resurgence of the deer population in the northeast United States and the incursion of suburban developments into rural areas where deer and black-legged ticks are commonly found may have contributed to the rising prevalence of human Lyme disease in the United States (Wilson 2002, Smith et al. 2001) and elsewhere (de Mik et al. 1997). Despite a similar increase in the deer population and in suburban development, locally acquired human cases of Lyme disease remain uncommon in Ontario. Climatic factors are known to limit tick populations and may contribute partly to the lower prevalence of the disease in Canada. However, other factors must be involved, since areas of the United States with generally similar climate to southern Ontario experience a much heavier burden of infection (Lindsay 2002, pers. comm.). Work by Schmidt and Ostfeld (2001) suggests that greater diversity of hosts for the tick may reduce infection rates by diluting the ticks' blood meals with incompetent hosts. Such diversity of mammalian hosts may contribute in part to the rarity of Lyme disease in Ontario relative to endemic areas in the neighbouring United States, though there is insufficient evidence at this time to assess the limiting role of host biodiversity in the distribution and prevalence of Lyme disease in the province.

Climate change may alter local conditions for ticks, the ecology and biodiversity of tick host species, bird migration timing and pathways, and the length of the transmission season in Ontario. Conditions that enhance the range and size of the tick population and that favor the transmission of *B. burgdorferi* in the rodent host population may increase the probability of human exposure to infected ticks. Continuing suburban developments in favourable tick and deer habitat, recreational activity in wooded areas, and exposure from pets will mean continued exposure to Lyme disease.

3.1.3. Rickettsial Diseases

Rickettsiae are very small intracellular bacteria. Several tickborne rickettsial diseases can occur in Ontario: Rocky Mountain spotted fever (RMSF, *Rickettsia rickettsii*), human granulocytic ehrlichiosis (HGE, *Anaplasma phagocytophila*), and Q fever, caused by *Coxiella burnetii*.

Tickborne typhus or RMSF is maintained in the wild in a number of tick species and vertebrate hosts. The principal vector in Ontario is the dog tick (*Dermacentor variabilis*). The disease is characterized by sudden onset of moderate to high fever and flu-like symptoms persisting two to three weeks. The organism has an affinity for the cells lining small blood vessels, hence the characteristic pimple-like rash that appears on extremities by the third day and spreads rapidly, with frequent occurrence of hemorrhages. If untreated, 15-20% of sufferers may die, but mortality is rare when antibiotic treatment is timely. Transmission is by bite of an infected tick and the incubation period ranges from 3 to 14 days (Walker and Raoult 2000). The prevalence of RMSF in Ontario is not well described, although the disease has been reported here (CDC 2002f, electronic access).

Human granulocytic ehrlichiosis (HGE) is a newly emerging disease. The vector is the black-legged tick (*I. scapularis*). The disease is similar to other manifestations of ehrlichiosis, with onset about 5-10 days after the tick bite of flu-like symptoms, possibly with vomiting, diarrhea, cough, joint pains, and confusion. A rash is almost never associated with HGE. Mortality is rare if the disease is treated in a timely manner, unless the patient has an already compromised immune system. Recently, the HGE agent, *Anaplasma phagocytophila*, was identified in a black-legged tick collected at Long Point, Ontario (Drebot et al. 2001). To date, no locally acquired human cases have been reported in Ontario (Artsob 2002, pers. comm.).

Q fever, another rickettsial illness, generally causes a mild flu-like syndrome. Severe complications including hepatitis may occur. The organism responsible for Q fever, *Coxiella burnetii*, is occasionally tickborne. The reservoir for *C. burnetii* is cattle (*Bovis domesticus*), sheep (*Ovis aries*), and goats (*Capra hircus*), with infection of herds common in Ontario (Lang et al. 1991, Lang 1989). Human cases of Q fever are generally acquired through direct exposure to infected animal tissues or by consumption of contaminated non-pasteurized milk. Human cases from tick bites are rare (Maurin and Raoult 1999). The organism can be transported long distances on dust particles, and windborne outbreaks have been documented (Hawker et al. 1998, Wellock 1960). Human cases related to dust blown off infected pastures might occur in Ontario if conditions favourable to outbreaks in livestock accompanied drought conditions and strong winds.

Although not a rickettsial disease, *Babesia microti* is a protozoan blood parasite also transmitted by the black-legged tick. Babesiosis and HGE are emerging diseases in the northeast and upper midwest regions of the United States and share the same vertebrate reservoir host (white-footed mouse [*P. leucopus*]) as the Lyme agent, *Borrelia burgdorferi*. There appears to be some risk for co-infection with any combination of these three organisms, plus the deer-tick virus (DTV) in Lyme disease-endemic areas (Varde et al. 1998); co-infection of ticks with multiple pathogens can occur.

The black-legged tick is a competent vector of many diseases. Clearly, any influence of climate on this tick and the dog tick and their host mammal populations could affect transmission dynamics of several relatively rare diseases and increase their public health importance. Ticks and their wildlife hosts are influenced by: land use and cover; soil type; elevation; and the timing, duration and rate of change of temperature and moisture regimes. Although tick survival and reproductive success appear to be most affected by microclimate (conditions in an individual tick's immediate environment), increased ambient temperature may accelerate development of pathogens within the tick, prolong the transmission season, and enhance over-wintering of infected ticks (Lindsay et al. 1995). Populations of reservoir mice, deer and other mammals could expand due to increased food availability and decreased winter mortality. Changes in human behaviour that might alter risk of exposure can be expected under climate change. For example, longer summers prolong the period of human exposure because of a longer summer recreational season.

3.1.4. Tularemia

Tularemia is caused by the bacterium *Francisella tularensis.* It is a zoonotic disease of rodents and lagomorphs and may be transmitted by the bites of deerflies, fleas, lice, or ticks, or by exposure to contaminated animal products (skins, meat). The disease was first identified after the 1911 San Francisco earthquake and was described as "plague-like" due to the similarity of symptoms. Tularemia has three clinical forms in humans: an ulceroglandular form with disease of the skin and lymph node enlargement resembling bubonic plague; an oculoglandular form, causing conjunctivitis and related lymph node involvement; and, a pneumonic form with severe pulmonary infection contracted by inhalation of the bacteria or dissemination by blood. Meningitis is a rare occurrence. All forms are accompanied by varying fever, aches and malaise. Effective antibiotic treatment is available. Complications of untreated disease include septicemia, kidney failure, and death (Cross and Penn 2000).

Recent concerns regarding bio-terrorism have lead to renewed interest and monitoring of the disease in the United States and elsewhere (Kaufmann et al. 1997). The distribution of tularemia is limited to the Northern Hemisphere, between 30 and 71 degrees latitude. Sporadic cases of the disease follow a seasonal trend, with a peak in summer associated with tick transmission and another peak in late winter associated with hunters. Thirteen species of tick are capable of transmitting tularemia, but the dog tick and wood tick are the main vectors of concern in Ontario. The disease has also been transmitted by deerfly (*Chrysops* spp.) bites.

Tularemia is not nationally reportable in Canada and is rarely reported in the United States. During 2000, 142 cases of tularemia were reported in the United States (CDC-DVID 2002h, electronic access). The incidence of tularemia in the United States has declined substantially since the 1950s. The disease occurs most frequently in the Midwest and on the island of Martha's Vineyard, Massachusetts. In those regions, children, the elderly, males, and Aboriginal people (probably due to greater prevalence of hunting) were more likely to acquire the disease (CDC 2002h, electronic access).

The prevalence of tularemia in Ontario wildlife and vector arthropods is not well described. Although climate change may affect the distribution of infected rodents and rabbits and the effectiveness of tick and deerfly vectors in Ontario, there is insufficient evidence to assess the impact of such changes on the prevalence of disease in humans. If climate change dramatically enhances the infection pressure in wild animal reservoir populations, human cases could ensue. Targeted surveillance of outdoor workers, hunters and trappers would permit the detection of changes in the ecology of tularemia in Ontario.

3.1.5. Exotic Vectorborne Diseases

a) Dengue

Dengue, a mosquito-borne disease caused by any one of four distinct but related serotypes of flavivirus, has become the second most important tropical disease worldwide, after malaria (WHO 2002a, electronic access). Infection is by bite of infectious mosquitoes (mainly *Aedes aegypti,* exotic to Canada) that prefer to feed during the day. Dengue fever is an acute febrile disease characterized by the sudden onset of fever lasting several days with an intense headache and other severe flu-like symptoms, sometimes including encephalitis. Cases usually require supportive care, and symptoms tend to resolve in time. The incubation period for the disease is approximately 3-14 days. Certain strains of virus may cause Dengue hemorrhagic fever (DHF), a serious condition with a fatality rate as high as 50%. DHF may occur more

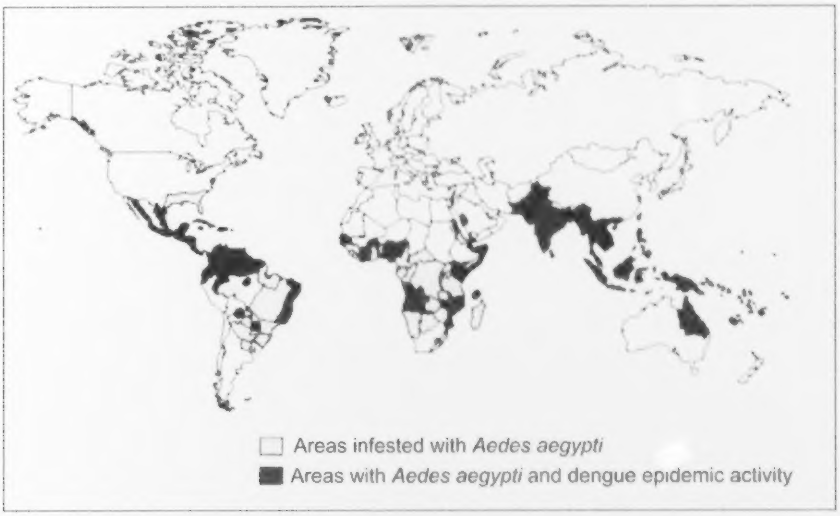

☐ Areas infested with *Aedes aegypti*
■ Areas with *Aedes aegypti* and dengue epidemic activity

Figure B.1. *World distribution of dengue, 2000. Dark gray areas are infested with the vector* Ae. aegypti *and have epidemic dengue. Light gray areas are infested with the vector; but do not have epidemic dengue. Courtesy of Center for Disease Control (CDC-DVBID, 2002c, electronic access).*

frequently in those with underlying illness or following a second exposure to Dengue. Dengue virus is maintained in primarily human-mosquito-human cycles. There is no specific treatment or vaccine available (Tsai 2000).

Dengue has experienced rapid global spread since the end of World War II, with an estimated 2.5 billion people now living in Dengue epidemic areas (Figure B.1). Mosquito eradication programs aimed at reducing yellow fever in the Americas successfully eliminated *Ae. aegypti* from most of the United States and Latin America by the late 1960s, preventing widespread Dengue. The eradication programs were discontinued, and the mosquito re-appeared, eventually obtaining a wider geographic distribution than before the eradication programs and allowing the spread of Dengue throughout the Americas. Recent outbreaks of Dengue and DHF in the Caribbean, Central and South America, and Mexico are of concern, as populations in the Americas are susceptible to infection, and the vector is present in the southern United States (CDC 2002c, electronic access). Increased international air travel and immigration contribute to possible future dengue transmission in the continental United States (Fayer 2000).

An additional concern is the spread of the mosquito *Aedes albopictus*, which was inadvertently introduced into the southern United States in the early 1980s (EHJ 2000, electronic access). It has subsequently spread throughout the southeastern USA. This mosquito is an effective bridge vector (feeds on birds and mammals) and can transmit Dengue and many other arboviruses of public health importance including Cache Valley, EEE and WNV (Moore and Mitchell 1997, Mellor and Leake 2000). Recent northward movement of *Ae. albopictus* as far as Minneapolis along the Mississippi River valley has been attributed to migratory birds (WHO 2002a, electronic access). Since 1980, three small outbreaks of locally transmitted Dengue have been reported in Texas near the Mexico border (CDC 1996). However, recent findings suggest a substantial level of under-reporting of Dengue cases (CDC 2001a).

Given increasing urbanization in developing countries, unavailability of safe pesticides, lack of effective mosquito breeding site eradication, and, as yet, the absence of an effective vaccine, climate change will likely enhance the spread of Dengue worldwide. Given the spread of the vectors in the United States, potential for travel-related cases in Canada is already increasing and will likely continue to grow. Since southern Ontario provides important resting places for migratory birds, it is possible that *Ae. albopictus* could be deposited here (CDC 2001a). For such vector introductions to be problematic, they would need to lead to the establishment of reproducing populations of mosquitoes. Climate would certainly play a role, as would habitat and preferred host availability. In addition, for human cases to occur in Ontario, some of the introduced *Ae. albopictus* would have to be infected with Dengue virus, and climatic conditions would need to be appropriate for transmission. At present, there is insufficient information to assess the likelihood of such a Dengue scenario. Improvements to passive surveillance systems should be made to allow the timely detection of new Dengue foci in North America and to better understand implications for Ontarians.

b) Malaria

Malaria is a well-described disease caused by infection with the protozoan red blood cell parasites *Plasmodium* spp., which are transmitted by various species of *Anopheles* mosquito. The disease is characterized by recurring attacks of chills and fever, which may become chronic. Malaria is distributed worldwide and is a major global health burden, with 40% of the world's population at risk of the disease (CDC 1990). Malaria causes more than 300 million acute cases of disease and at least one million deaths annually. Ninety percent of deaths due to malaria occur in sub-Saharan Africa, and most involve young children (WHO 2002d, electronic access). Although it is now confined to tropical regions of the globe, malaria was endemic in marshy areas of southern Ontario and Québec and throughout much of the United States in the 17th to early 20th centuries (Zucker 1996).

Interrupted human-vector contact, decreased *Anopheles* mosquito populations, and effective treatment contributed to a decline in transmissions and to subsequent eradication in Ontario. However, environmental changes, the spread of drug resistance and increasing air travel could lead to the re-emergence of malaria as a public health problem in Ontario. Southern Ontario is already in a climatic zone where malaria transmission could occur. With climate change, this area would expand to include more of the province (HC-SAB 2000, electronic access).

Numerous limited outbreaks of imported malaria have occurred in the United States (California, Georgia, Florida, Louisiana, Maryland, also New York, New Jersey) (CDC 1982, 1986, 1990, 1991, 1997b, 2002). These outbreaks result when a person carrying *Plasmodium* arrives in the United States and is then bitten by local *Anopheles* mosquitoes. The now-infected mosquito can then spread the disease (Zucker 1996). Local transmission of malaria is present and increasing in Moscow, formerly considered too cold for *Anopheles*. However, the mosquito was found in most bodies of water in the Russian capital (ProMed 2002, electronic access). A single case of locally acquired malaria may have occurred in Scarborough in 1998 (Keystone 2002, pers. comm.).

The time required for the complete maturation of the parasite in the mosquito varies, depending on the *Plasmodium* species and external temperature. A temperature relationship for sporadic local malaria transmission was observed in New York and New Jersey during the 1990s; common to these outbreaks was exceptionally hot and humid weather that reduced the development time of malaria sporozoites sufficiently to render northern *Anopheles* mosquitoes infectious (Layton et al. 1995, Zucker 1996). Two cases of locally transmitted malaria were reported in 2000 in Suffolk County, New York (Bradley et al. 2000). The cases occurred shortly after a heavy rainfall during one of the warmest and driest summers in history. Dry weather followed by heavy rains may result in conditions conducive for mosquito breeding and reduce mosquito predator populations. Despite numerous past occurrences of climate conditions favorable to the local transmission of malaria, outbreaks have been rare.

Factors limiting malaria transmission in Ontario today include a low proportion of infected people and the relatively small proportion of *Anopheles* mosquitoes in the mosquito population. Several factors affect mosquito distribution, of which climate may not play the most important role (Reiter 2000). Of the 74 species (12 genera) of mosquito found in Canada, six belong to the genus *Anopheles* (Lindsay 2002, pers. comm.). The actual prevalence of *Anopheles* mosquitoes in Canada is unknown. However, an increase in this proportion due to climate change would increase the likelihood of the occurrence of locally transmitted imported malaria. Trends in immigration from, and travel to, malaria-endemic locations also affect the likelihood of malaria transmission in Ontario.

In tropical countries, most malaria is transmitted at relatively stable rates in areas where the disease is endemic. Malaria epidemics do occur, precipitated by factors in two categories: natural (climatic variations, natural disasters) and man-made (conflict and war, agricultural projects, dams, mining, logging). Most of these factors modify the physical environment, increasing the capacity of mosquitoes to transmit malaria. The lack of public health services and infrastructure can also trigger outbreaks. Some factors also result in vast human population movements that expose non-immune populations to malaria infection. Because malaria epidemics occur in populations not normally exposed to the disease, or who are exposed for only a short part of the year, local health services are usually unprepared to predict, detect and control such epidemics in time, resulting in severe cases and high death rates. Although explosive epidemics of malaria in Ontario are unlikely to occur, the population is highly susceptible to infection, and our public health system is unprepared to recognize or respond to it. Malaria is a serious disease, and any increase in the number of cases in Ontario would be of concern. Efforts to understand the importance of climatic, geographical, social and other factors in recent North American outbreaks of malaria will help public health professionals in Ontario understand the potential implications of climate change on malaria in the province.

In addition to local or continental changes in disease distribution, climate change-related alterations in the range and prevalence of various important vectorborne diseases worldwide could significantly affect on the health of Ontario residents who travel outside of the country. Malaria is now the most prevalent vectorborne disease worldwide. Global increases in endemic malaria, increased resistance to anti-malarial drug therapy, and a significant increase in global travel have resulted in thousands of cases of malaria transported into Europe and North America annually, a few giving rise to transmission by indigenous mosquitoes (Fayer 2000). Ontario consistently has the highest incidence of imported cases of malaria, likely due to its population size and Toronto's importance as a travel and immigration centre. Annual incidence has been as high as 444 cases (1997) (HC-PPHB 2002a, electronic access), though only 131 cases were reported in 2001 (Health Canada 2002a).

3.2. Foodborne and Waterborne Infections

Food and water are vehicles for a great number of human infections and parasitic infestations. Many of these infections cause gastroenteritis (inflammation of the stomach and intestine), with symptoms of diarrhea and vomiting. Foodborne and waterborne illnesses are perhaps the most important global health problem. Of the approximately 1.5 billion cases of diarrhea occurring annually worldwide and causing the deaths of 3 million children under five, 70% have been estimated to have been caused by biologically contaminated food (WHO 2002b, electronic access). In Ontario, as in other wealthy industrialized nations, good sanitation and refrigeration, public health and food safety programs, and effective treatment of drinking water provide important defenses against foodborne and waterborne diseases. Nonetheless, many outbreaks still occur, some affecting hundreds of people (HC-PPHB 2002a, electronic access). Many enteric infections are known or suspected to be foodborne or waterborne, and many more cases are not reported (Notermans and Borgdorff 1997). Although most enteric pathogens can be either foodborne or waterborne, each pathogen tends to be transmitted in a specific vehicle. In addition, some non-enteric pathogens are food or waterborne, including *Toxoplasma gondii*, *Listeria monocytogenes*, and *Leptospira*, which cause severe illnesses affecting major organ systems (brain, liver, kidney, heart). Most foodborne and waterborne pathogens are zoonotic, with reservoirs in livestock and wildlife, from which they spread to water and an increasing array of foods.

Climate change may alter the ecology of many foodborne and waterborne diseases in Ontario. Warmer summer temperatures are expected to enhance microbial survival and replication in food and water, thus increasing the risk of infection. Warmer temperatures will also increase the strain on refrigeration systems, causing more frequent food spoilage. Microbial ecology on farms may change with altered climate, potentially changing the species composition of pathogens and their infectivity to people. Water contamination events will be affected by the rates and timing of precipitation events and by water demand. The complexity of the interactions among environmental factors, reservoir hosts, and pathogen ecology in the epidemiology of foodborne and waterborne diseases suggests a likely impact of climate change on the occurrence of these diseases in Ontario. However, the burden of illness from foodborne and waterborne pathogens is rarely well-described and is believed to be widely underestimated. Thus, estimating the net impact of climate change on foodborne and waterborne infections in Ontario is difficult.

3.2.1. Foodborne Infections

Foodborne diseases display a strong summer seasonal peak in Canada and other industrialized countries with a temperate climate (Isaacs et al. 1998, Bentham and Langford 2001). This seasonal pattern is attributed to warmer weather affecting rates of microbial replication, food spoilage, and food preparation and consumption behaviour. Foodborne diseases appear to be on the rise (Notermans and Borgdorff 1997), with the emergence of several previously unrecognized pathogens and a resurgence of well-known enteric pathogens (such as *Salmonella*). These changes may be linked to a combination of factors, such as environmental change (including climate), antimicrobial drug use, transportation of pathogens, human movement and behaviour, and the globalization and intensification of food production. The potential for large-scale epidemics is exacerbated by globally consolidated food processing and distribution networks (WHO 2002c, electronic access). The severity of foodborne infections is also changing, as pathogens acquire greater genetic virulence and resistance. The influence of climate change on these trends in foodborne illness has not been widely studied.

Every year in Canada, foodborne infections cause significant levels of illness and some deaths, but the actual incidence is not known (Khakhria et al. 1997). Most of these infections cause only minor gastrointestinal illness. Many infections escape the reporting system since the patients do not seek medical care and are not tested by a laboratory. Even when laboratory tests are conducted, the source is often not identified. Some cases of gastrointestinal illness are linked to outbreaks (several cases from a common source), but most occur sporadically as isolated occurrences, making source identification difficult.

Four years of data on nationally reported outbreaks of foodborne, waterborne, or enteric diseases are summarized in Table B.2. Ontario routinely reports 9-15 outbreaks of foodborne, waterborne or enteric illness annually. Although the number of outbreaks is not excessive relative to other provinces, the number of people affected has been very large, in some instances over 2,500. This means that Ontarians were affected more than other Canadians between 1997 and 2000. By comparison, there were 370 outbreaks reported in Canada over the four years, affecting 8,056 people. The source was identified in only 74 of those outbreaks; most were foodborne (Health Canada 1999, 2002b).

Since the reporting structure is based on laboratory diagnosis, the causative agent of nationally reported outbreaks is usually known (Table B.3). Bacteria are the most important source of foodborne enteric illness in

Table B.2. *Outbreaks of foodborne, waterborne or enteric disease for Ontario and Canada from 1997-2000 (Health Canada 2002b). The numbers of outbreaks and cases in Ontario are ranked nationally. The fourth column gives the number of outbreaks of known source that were foodborne in Canada.*

Year	Ontario outbreaks (cases)	Rank outbreaks (cases)	Canada outbreaks (cases)	Foodborne outbreaks (Total known)
1997	12 (69)	4 (5)	97 (1355)	16 (19)
1998	14 (586)	2 (1)	75 (2248)	16 (20)
1999	9 (250)	3 (1)	55 (919)	15 (18)
2000	13 (1720)	4 (1)	143 (3534)	16 (17)
TOTAL	48 (2625)		370 (8056)	63 (74)

Canada. Of the 344 outbreaks in Canada caused by primarily foodborne agents between 1997 and 2000, more than one third (123) were due to *Salmonella* (Health Canada 1999, 2002b), similar to what has been reported in the United States (Meade et al. 1999). A large number (86) were attributed to Norovirus (Norwalk virus and Norwalk-like caliciviruses) with most of these occurring in 2000. Verotoxigenic *E. coli* infection was responsible for 20% (66) of the outbreaks, and *Shigella* for just under 10% (32 outbreaks). No other agent was responsible for more than 3% of outbreaks (Health Canada 1999, 2002b).

Food may be contaminated anywhere along the production chain (Figure B.2). The dynamic complexity of food and water systems is such that feedback loops within these systems may result in unexpected effects of pathogens or unexpected outcomes of mitigation strategies. Elevated temperatures projected for Ontario in the coming decades may exacerbate the problem of pathogen contamination during livestock rearing, food processing, and food preparation. Outbreaks of zoonotic diarrhea in heat-stressed livestock may be an additional source of contamination. Sustained hot temperatures for long periods may result in more frequent lapses in refrigeration during transport or storage and in the home, thus allowing microbial growth in foods.

Climate change will likely increase the frequency of heat waves in Ontario, with associated poor air quality (smog). People who have underlying respiratory and cardiovascular illnesses are vulnerable to an exacerbation of their conditions during heat waves. Such people might be more vulnerable to gastrointestinal illness as well (elderly residents of nursing homes, for example), and in the event of infection be subjected to more severe symptoms of either or both their underlying condition and the gastrointestinal problem. Add to these issues the problem of antibiotic resistance, and the implications for public health are quite serious (Hosek et al. 1997, Gaudreau and Gilbert 1998).

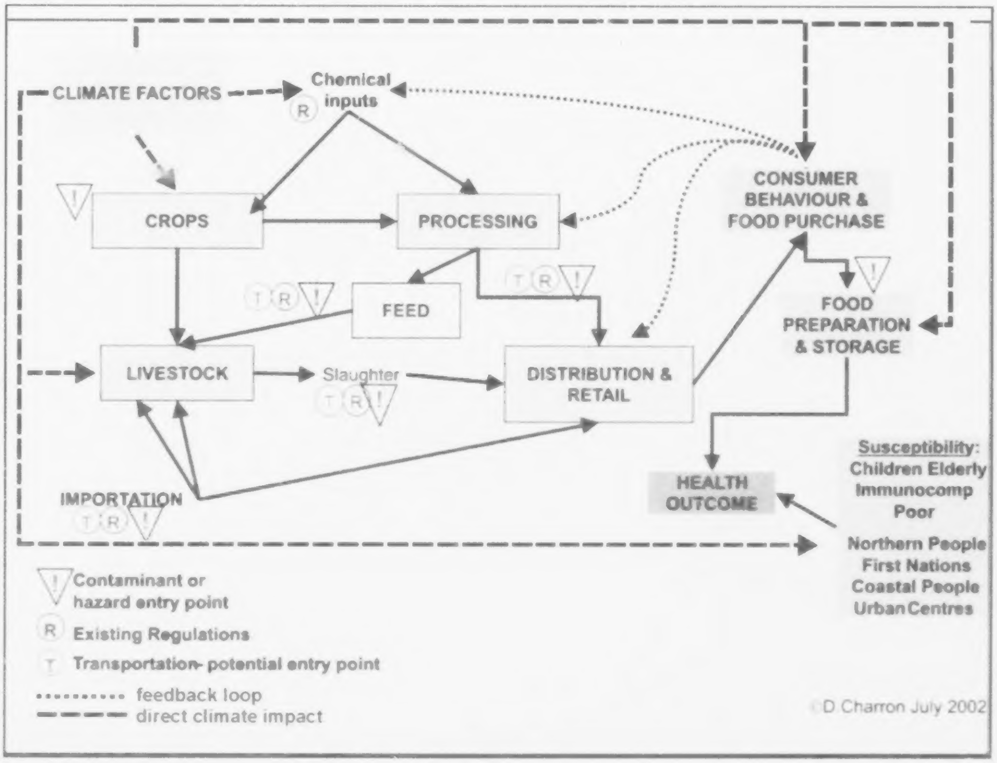
Table B.3. *Number of outbreaks of foodborne, waterborne, or enteric disease in Canada by causative agent, 1997-2000 (Health Canada 1999, Health Canada 2002b). Pathogens known to be primarily waterborne were omitted from this table.*

Causative agent	1997	1998	1999	2000	TOTAL
Salmonella	43	26	17	37	123
Verotoxigenic Escherichia coli (including O157:H7)	20	15	14	17	66
Campylobacter jejuni/coli	1	2	2	3	8
Clostridium botulinum	3	-	-	-	3
Clostridium perfringen	1	-	1	2	4
Shigella	8	9	6	9	32
Vibrio parahaemolyticus	1	-	-	-	1
Bacillus cereus	-	-	-	1	1
Yersinia enterocolitica	-	1	1	-	2
Norovirus (Norwalk virus, Norwalk-like and other calicivirus)	6	11	7	62	86
Rotavirus	-	1	2	4	7
Hepatitis A	3	1	-	-	4
Cyclospora	1	1	2	-	4
Paralytic shellfish Poisoning	-	2	-	-	2
Ciguatera	-	1	-	-	1
TOTAL	**87**	**70**	**52**	**135**	**344**

Climate change may also change food consumption and preparation patterns in Ontario. Outdoor grilling is popular in the warmer months and is often linked to outbreaks of foodborne illness from undercooked ground beef (Todd 2000). What's more, the availability of fresh vegetables and fruits in the summer results in more consumption of these foods. Produce that has been contaminated with fecal matter (sewage or manure) can be a significant source of foodborne illness if it is not washed properly. Food consumed in Ontario often comes from outside the province. Pathogen levels in food grown elsewhere will have been influenced not only by the climate at source, but by environmental conditions during transport and processing.

Through social, cultural, and technological changes linked to climate, changes may occur in food quality and availability worldwide. Changes in the prevalence of malnutrition and of starvation, in turn, may contribute the movement of human populations. These situations are often favourable to disease spread (Martens 1999) and may alter the epidemiology of diseases endemic to those areas and diseases with global distribution. Although food security is less likely to be affected in Ontario and other North American jurisdictions, socio-cultural impacts of climate change may include changes in type of employment, time spent outdoors, recreational activities, and food storage and preparation that may increase the risk of enteric and other diseases.

Many Aboriginal and other northern residents of Ontario who undertake hunting, fishing, and other resource-based activities for subsistence may be more vulnerable to the influence of climate change on foodborne illness. These groups pose a particular research and action challenge, since such foods are not regulated. Climate change could also alter the abundance and distribution of wildlife, fish, and vegetation (IPCC 2001). As a result, food supplies and economic livelihoods of many First Nations peoples could be in jeopardy (Last et al. 1998, Weller and Lange 1999). Disappearance of traditional food and medicinal plants from areas populated by Native American and other indigenous peoples may likewise affect physical, mental, and spiritual well being (IPCC 2001).


CLIMATE FACTORS
Chemical inputs
R
CROPS
PROCESSING
CONSUMER BEHAVIOUR & FOOD PURCHASE
!
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FEED
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FOOD PREPARATION & STORAGE
LIVESTOCK
Slaughter
T R !
DISTRIBUTION & RETAIL
Susceptibility:
Children Elderly
Immunocomp
Poor
HEALTH OUTCOME
IMPORTATION
T R !
Northern People
First Nations
Coastal People
Urban Centres
! Contaminant or hazard entry point
R Existing Regulations
T Transportation- potential entry point
feedback loop
direct climate impact
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Figure B.2. *Influence of climate on food production systems and hazard entry points, regulatory nodes, and transportation issues.*

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Through social, cultural, and technological changes linked to climate, changes may occur in food quality and availability worldwide. Changes in the prevalence of malnutrition and of starvation, in turn, may contribute the movement of human populations. These situations are often favourable to disease spread (Martens 1999) and may alter the epidemiology of diseases endemic to those areas and diseases with global distribution. Although food security is less likely to be affected in Ontario and other North American jurisdictions, socio-cultural impacts of climate change may include changes in type of employment, time spent outdoors, recreational activities, and food storage and preparation that may increase the risk of enteric and other diseases.

Many Aboriginal and other northern residents of Ontario who undertake hunting, fishing, and other resource-based activities for subsistence may be more vulnerable to the influence of climate change on foodborne illness. These groups pose a particular research and action challenge, since such foods are not regulated. Climate change could also alter the abundance and distribution of wildlife, fish, and vegetation (IPCC 2001). As a result, food supplies and economic livelihoods of many First Nations peoples could be in jeopardy (Last et al. 1998, Weller and Lange 1999). Disappearance of traditional food and medicinal plants from areas populated by Native American and other indigenous peoples may likewise affect physical, mental, and spiritual well being (IPCC 2001).

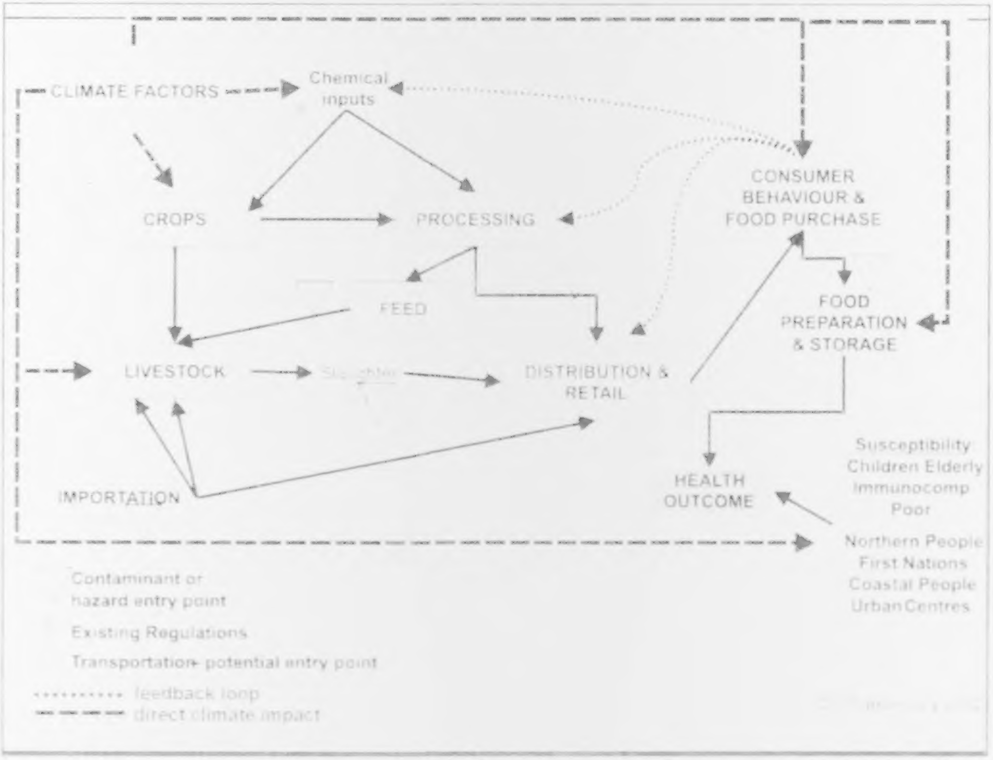
CLIMATE FACTORS
Chemical inputs
CROPS
PROCESSING
CONSUMER BEHAVIOUR & FOOD PURCHASE
FEED
FOOD PREPARATION & STORAGE
LIVESTOCK
Slaughter
DISTRIBUTION & RETAIL
IMPORTATION
HEALTH OUTCOME
Susceptibility:
Children Elderly
Immunocomp
Poor
Northern People
First Nations
Coastal People
Urban Centres
Contaminant or hazard entry point
Existing Regulations
Transportation- potential entry point
feedback loop
direct climate impact

Figure B.2.

a) Foodborne Enteric Pathogens

The most common foodborne diseases are caused by enteric pathogens, including *Salmonella* and *Campylobacter*, Noroviruses and other enteric viruses. For most enteric pathogens to cause illness, the contaminated food must be eaten raw, inadequately cooked, or contaminated after cooking. The pathogens cause illness by multiplying in the intestine of the host. Foodborne intoxications (from the toxins produced by *Clostridium botulinum*, *Staphylococcus aureus* or *Bacillus cereus*) are less commonly isolated from human cases since these toxins have a short half-life in the digestive tract. These toxins are produced by the bacteria as they multiply in the food (or the intestine in the case of *B. cereus*), and the toxins cause the symptoms. Outbreaks of botulism occasionally occur among Canada's Aboriginal communities, usually in association with ill-preserved game meat (Health Canada 1999).

E. coli O157:H7 and other verotoxigenic *E. coli* cause a severe intestinal infection that generally results in bloody diarrhea, vomiting, fever, and can lead to complications such as hemolytic uremic syndrome, permanent kidney failure, and death (Slutsker et al. 1997). Children and the elderly are particularly vulnerable to this infection. Foodborne *E. coli* O157:H7 is becoming more common, with outbreaks linked to undercooked ground beef (Slutsker et al. 1998) and raw sweet apple cider (Hilborn et al. 2000).

In affluent, industrialized locales like Ontario, foodborne enteric illness is usually mild and self-limiting, rarely resulting in death. Yet, because so many people are affected each year, the economic burden associated with illness is very high. In Canada, the annual cost of foodborne disease (2.2 million cases caused by 13 etiologic agents) has been estimated at $1.3 billion in 1985 dollars (Buzby and Roberts 1997, Todd 1989).

Figure B.3 shows the number of cases of campylobacteriosis, salmonellosis, shigellosis and verotoxigenic *E. coli* (VTEC) reported from Ontario between 1992 and 2001. On average, about 9,000 people are infected with one of these four pathogens annually, causing economic losses estimated at over $5 million (in 1985 dollars) (based on Buzby and Roberts 1997).

Livestock and other food animals are reservoirs for many disease-causing organisms, including *Salmonella*, verotoxigenic *E. coli*, *Campylobacter*, and *Yersinia enterocolitica* (for example, Khakhria et al. 1997, Van Donkersgoed et al. 1999, Letellier et al. 1999, Mikaelian et al. 1997). Food may also become contaminated by people infected with these and other pathogens, such as *Shigella*. Pathogens may also enter food that is grown, washed or prepared using contaminated water (*Cyclospora cayatenensis* in berries, for example).

A recent report from the U.S. Centers for Disease Control describes a decline in the incidence of several major foodborne infections in foodborne disease surveillance (Foodnet) locations over the past few years (CDC 2002a). Exceptions included *Salmonella* enteritidis, *Salmonella* Heidelberg, *Vibrio*, *E. coli* O157, and *Shigella*. The decline was attributed to the implementation of new control measures such as enhanced food inspection and Hazard Analysis Critical Control Point (HACCP) systems in the food industry, including egg production, fresh produce, and seafood. The report underlines that the overall incidence of foodborne disease remains high. Areas of concern include reservoirs of *E. coli* O157 (cattle), *Salmonella* enteritidis (laying hens), and *Vibrio* (oysters); the rise in new serovars of *Salmonella*; and the elevated incidence of foodborne illness among infants and children.

Most enteric infections acquired in Canada and the United States exhibit a pronounced seasonal pattern, with a peak in the summer (Isaacs et al. 1998). A rise in enteric infections generally occurs in February and March due to infections acquired by Canadians vacationing abroad. Generally, warmer temperatures favour the

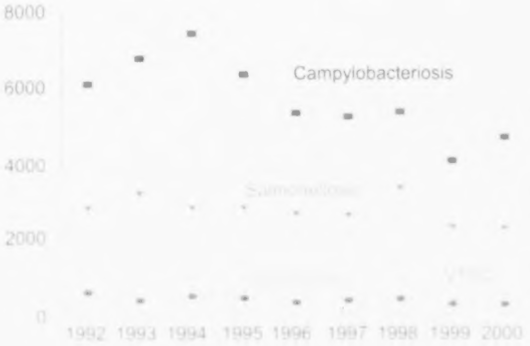

Figure B.3. *Number of cases of campylobacteriosis, salmonellosis, shigellosis and verotoxigenic* E. coli *(VTEC) infection from Ontario reported federally from 1992 to 2001.*

contamination of food during production and preparation, due to increased replication and survival of pathogens in food, lapses in refrigeration, and perhaps higher incidence in animal reservoirs (Bentham and Langford 2001). Therefore, the potential for increased foodborne illness with climate change has been suggested (Bentham and Langford 1995). Preliminary data from Alberta and Newfoundland suggest that the warming trend over the 1990s is mirrored by an upward trend in the incidence of major foodborne pathogens (Figure B.4, Health Canada 2002b).

Precipitation levels may affect the availability or quality of water available for irrigation and food processing, providing the opportunity for food contamination at the farm or in the processing plant. Changes in the ecology of diseases in livestock may occur because of climate change, resulting in the emergence of new pathogens and in changes in antimicrobial use, which may in turn alter the risk of human illness.

b) Hepatitis A Virus

Hepatitis A is caused by a picornavirus. Surveys conducted in industrialized countries (Japan, United States, Australia, Europe) reveal that 40-70% of adults have antibodies to the hepatitis A virus (HAV) (Feinstone and Gust 1999). Symptoms of hepatitis A infection generally resemble the flu and may progress to severe abdominal pain and jaundice, symptoms that cannot be distinguished from other types of acute hepatitis. Unlike Hepatitis B and C (which are bloodborne), HAV infection does not lead to lifelong infection. The disease is generally transmitted via the fecal-oral route, either directly through interpersonal contact or indirectly through ingestion of contaminated food or water (often in outbreaks) (Wu et al. 2001). In North America, household or sexual exposure is far more commonly identified than food or water as the potential source of infection (Gully and Tepper 1997, Zou et al. 2000). Travel to countries where hepatitis A is endemic is an important risk factor for acquiring the disease. The reported incidence of hepatitis A in Ontario is between 250-600 cases annually, with a peak of over 1,000 in 1991, declining in recent years (HC-PPHB 2002a, electronic access). A study of the sero-prevalence of the disease in a young, urban, Canadian-born population suggests a high level of susceptibility and thus risk of outbreak in this group (Levy et al. 2001). First Nations communities may be particularly susceptible to infection (Harb et al. 2000). Foodborne outbreaks have occurred in Ontario and elsewhere in Canada (Health Canada 1999). It is generally accepted that the true incidence of HAV infection is considerably underestimated because of under-reporting of clinical illness and the occurrence of sub-clinical infections in children (Wu et al. 2001). In Canada, hepatitis A vaccine is currently recommended by the National Advisory Committee for Immunization (NACI) for individuals at increased risk, including travelers and residents of communities with elevated levels of hepatitis A infection (Gully and Tepper 1997).

Hepatitis A may also be waterborne, and like other waterborne pathogens, it could pose an increased threat to vulnerable populations if climate change were to bring more frequent or heavier rains, flood or drought. Since Aboriginal Canadians may be at increased risk of the disease and may also be more vulnerable to climate change, Ontario's First Nations communities would benefit from enhanced surveillance and from vaccination where appropriate.

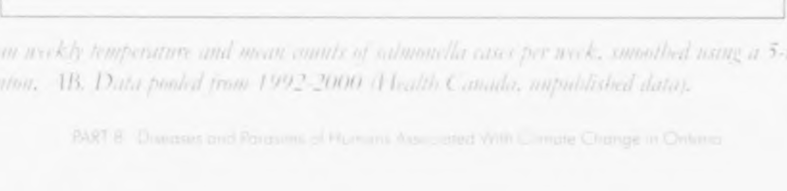

Figure B.4. *Mean weekly temperature and mean counts of salmonella cases per week, smoothed using a 5-week moving average, for Edmonton, AB. Data pooled from 1992-2000 (Health Canada, unpublished data).*

c) Listeriosis

Listeriosis is a rare disease caused by the bacterium *Listeria monocytogenes*. Recent outbreaks of listeriosis have been linked to food sources: processed cold turkey (CDC 2000a) and pasteurized cheese (HC-PPHB 2002a, electronic access). The organism is found in the environment and is carried asymptomatically by many mammals, including humans, and in birds, fish, and crustaceans. Cattle acquire this environmental pathogen from wet spoiled hay bales or improperly fermented silage, which generally develop following wet spring weather (Fenlon et al. 1996). Historical outbreaks of the disease have been associated with raw milk products and, more rarely, with uncooked refrigerated processed meats (CDC 1999). The organism is capable of proliferating in products refrigerated at 4°C (Lorber 1997).

Infection is generally asymptomatic in adults. In the elderly, newborns, and those with compromised immune function, severe meningoencephalitis and/or septicemia may occur. Infection during pregnancy may be transmitted to the fetus causing miscarriage, stillbirth or neonatal illness. The incubation period in adults varies but is usually around 3 weeks in duration. Clinical listeriosis occurs most often in infants (40% of all cases) and is fatal in up to half of all neonatal cases. Human cases of listeriosis are rarely reported in Ontario, between 20 and 50 cases per year since 1992 (HC-PPHB 2002b, electronic access).

The prevalence of listeriosis in Ontario is related to food choices and underlying susceptibility. Changes in climate may increase the potential for contamination of milk and meats with pathogen, but is unlikely to affect the overall incidence of disease in people.

d) Infections and Intoxications From Fish and Seafood

Fish and shellfish may be sources of pathogens and toxins that cause enteric disease or neurological symptoms. Outbreaks of illness linked to these foods are rarely reported in Ontario or nationally (Health Canada 1999, 2002b). Infections and intoxications from fish and seafood include ciguatera poisoning (most commonly caused by eating carnivorous tropical reef fish contaminated by a toxin produced by the dinoflagellate *Gambierdiscus toxicus*); paralytic (PSP), amnesic (ASP) and diarrheic (DSP) shellfish poisonings, also related to various algal or plankton blooms (Todd 1994, Morris 2000); *Vibrio parabaemolyticus* gastroenteritis from undercooked or raw shellfish (Health Canada 1997); and various environmental and fecal pathogens from eating shellfish grown in contaminated water.

Vibrio parabaemolyticus and other marine *Vibrio* multiply rapidly following plankton blooms (Neill and Carpenter 2000). Contamination of mollusks is linked to climate: In the warmer months, mollusks routinely acquire *Vibrio* as they filter-feed and not as a result of fecal contamination of waters.

In the United States, PSP is associated with eating mollusks. Approximately 10 outbreak-associated PSP cases are reported to the Centers for Disease Control and Prevention each year, whereas only two outbreaks of PSP have been reported in Canada between 1997 and 2000. Neurological symptoms of intoxication occur within 2 hours of ingestion and include tingling and burning of the mouth and tongue, numbness, and drowsiness. Large doses may be fatal. The toxins are produced by Gonyaulacoid dinoflagellates and by some freshwater cyanobacteria. Shellfish are contaminated by filter-feeding in waters affected by algal blooms where the toxins are produced in abundance. In general, fish and crustaceans are not affected. However, pufferfish feed on mollusks and might accumulate or even magnify the toxin (Health Canada, 2002a).

Algal blooms seem to be increasing in frequency, and climate change may be involved (Tester 1994). The ecology of estuaries and oceans will likely be profoundly affected by climate change (IPCC 2001, electronic access), with implications for algal bloom frequency and distribution. Ontarians who consume fish and shellfish are at risk of illness related to these blooms. Changes in climate may exacerbate the risk of acquiring such seafood-borne illnesses when travelling outside the province or from consuming contaminated products imported into Ontario.

3.2.2. Waterborne Infections

The risk of waterborne illness in Ontario could be affected by altered patterns of precipitation, floods (increased runoff, decreased effectiveness of treatment, combined sewer overflow), high temperatures (pathogen replication), and drought (through the concentration of pathogens in smaller volumes of water, decreased hygiene measures due to water shortages, decreased water pressure in treatment plants, etc.). There may be increased risk that heavy rain or snowmelt may flush manure, human sewage, and wildlife and pet droppings into surface drinking water reservoirs or ground water, leading to widespread contamination of drinking water sources.

Waterborne diseases are caused by pathogens spread through contaminated drinking water or exposure to contaminated water while swimming or doing other activities. For drinking water to be a source of illness, the water must become sufficiently contaminated and then escape treatment, or treatment must fail. Water may become contaminated by animal or human waste at the source: human sewage, leaking septic systems, manure runoff from agricultural lands, and wild animal wastes may all contaminate surface water later used for drinking water. Ground water may become contaminated by surface contamination of wells, sub-surface inflows, improperly situated septic fields, or leaking dumps (chemical contamination). Treated drinking water may become contaminated later. A persistent threat to Ontario public health is posed by antiquated combined sewer systems (CSS), which carry both storm water and raw sewage to the sewage treatment plant. When the flow of water is too great for the system (heavy rainfall, snowmelt, etc.), the sewers overflow directly into a surface water body (river, lake). Thus microbial pathogens, industrial wastes, and city street contaminants run untreated into water, contaminating downstream drinking water sources, beaches, and fish and shellfish. All CSS in Ontario predate 1956. More than 67 municipalities have CSS, providing service to millions of people. Seventy-five percent of large city residents are served by these systems (Totten Sims et al. 2001, electronic access).

Often, untreated drinking water is implicated in waterborne disease outbreaks. However, very large outbreaks may occur when municipal treatment systems fail: More than 400,000 people were affected in an outbreak in Milwaukee, Wisconsin (MacKenzie et al. 1994), while in Walkerton, Ontario, over 3,000 were affected (Bruce-Grey-Owen Sound Health Unit 2000). Some pathogens are particularly resistant to treatment, in particular chlorine-resistant protozoa like *Cryptosporidium* (Payment 1999).

Climate projections for Ontario may significantly affect drinking water quality. On the whole, Ontario will likely experience longer summers and milder, wetter winters, while the southwest part of the province will likely experience increased summer drought (NRCAN 2002, electronic access). In a report prepared for multiple conservation stakeholders and the Canadian Water Resources Association, regional projections were summarized for Ontario (Bruce *et al.*, 2000, electronic access). The projections included increased overall annual precipitation with reduced snow and increased rainfall, more extreme weather (thunderstorms) (see also Francis and Hengeveld 1998, Groisman and Easterling 1994), more surface runoff and flooding, and less infiltration. Meanwhile, overall increased temperature will promote evaporation, offsetting the increased precipitation and resulting in lowered lake levels. Increased precipitation frequency and intensity have been noted in recent years in some areas of North America (Karl et al. 1995), although this trend is not evident over the last 50 years in Ontario (Environment Canada, unpublished data).

a) Common Waterborne Enteric Pathogens

Many of the enteric pathogens described in the section on foodborne infections can be transmitted through contaminated water. People may also acquire infections from eating food that was prepared using contaminated water. The protozoan infections caused by *Giardia* spp. and *Cryptosporidium* are commonly transmitted in water in Ontario (Majowicz et al. 2000) and elsewhere (Table B.4). Although toxoplasmosis can be contracted by ingesting material contaminated with infected cat feces or consuming undercooked pork infested with *Toxoplasma* cysts, the disease has been associated with large waterborne outbreaks (Aramini et al. 1999).

Giardia lamblia is the most commonly reported cause of outbreaks of waterborne illness in Canada and the United States (Table B.4). In Canada, *Cryptosporidium* and *Campylobacter* account for most of the remaining outbreaks of illness where the source was confirmed to be water. Majowicz et al. (2000) found the mean incidence of cryptosporidiosis in Ontario in 1996-97 to be 2.13 per 100,000 people, but suspected substantial under-reporting of the disease due to sporadic testing for the pathogen. The study found that males, children under 5 years of age, and rural people were at elevated risk. Of the cases where a suspected source was reported, 48%

Table B.4. *Waterborne disease outbreaks of known cause associated with drinking water reported to the United States CDC, 1972-94 (Mead et al. 1999) and to Health Canada, 1975-2001. The total number of outbreaks is given in parentheses (Health Canada 2002b).*

Organism	USA (291) %	Canada (37)[1] %
Giardia lamblia	37.5	41
Cryptosporidium	3.4	22
Campylobacter jejuni	5.2	22
Salmonella	6.2	5
Shigella	11.7	3
E coli O157 :H7 (and other VTEC)		3
Yersinia enterocolitica		3
Hepatitis A	7.2	3
Norovirus	5.5	6
Rotavirus		3
Chemicals	21	

[1] 10 of these outbreaks were in Ontario

listed water; livestock exposure was a factor in 21% of cases, person-to-person transmission was a factor in 15% of cases, and travel outside of the province was a factor in 22% of cases. Nationally, other causes of waterborne disease outbreaks include Noroviruses, *Salmonella*, and other viruses and bacteria. In the United States, chemical contaminants, *Shigella*, hepatitis A, and *Salmonella* were also each responsible for more than 5% of the outbreaks.

Although the burden of waterborne illness in Ontario is unknown, the above data suggest that it is responsible for a significant proportion of enteric illness. Most of the 1841 cases of giardiasis and 200 cases of cryptosporidiosis reported in Ontario in 2001 (Health Canada, 2002a) were presumed waterborne. Payment et al. (1991) estimated that 35% of all enteric disease in Montreal was due to preventable waterborne illness. Like that of foodborne illness, the incidence of waterborne illness is under-reported since illness is usually self-limiting in healthy adults, and medical attention is not sought (Frost et al. 1996). However, severe disease can occur in all age groups. The large waterborne outbreak of *E. coli* O157:H7 in Walkerton, Ontario, in 2000 had severe consequences and brought demands for political action (McQuigge et al. 2000). Native peoples may be particularly at increased risk of waterborne illness due to inconsistencies in the availability of safe drinking water in remote areas (Rosenberg et al. 1997).

Livestock may be asymptomatic carriers of zoonotic pathogens that are shed in manure and can contaminate drinking water. Linkages between agricultural contamination and precipitation events have been noted in the United States (MacKenzie et al. 1994) and in Ontario (McQuigge et al. 2000). Over half the waterborne disease outbreaks in the United States in the latter half of the 20th century were preceded by an extreme precipitation event (Curriero et al. 2001).

Weather is often a factor in triggering waterborne disease outbreaks. Excess rainfall resulted in surface contamination of ground water and contributed to the Walkerton outbreak of *E. coli* O157:H7 (Auld et al. 2001). Drought followed by heavy rainfall preceded a large waterborne outbreak of *E. coli* O157:H7 in New York in 1999 (Patz et al. 2000). Extreme precipitation preceded the massive outbreak of *Cryptosporidium* in Milwaukee in 1993 (MacKenzie et al. 1994) and preceded several other outbreaks of waterborne illness (Rose et al. 2001). A large waterborne outbreak of toxoplasmosis in Victoria BC was associated with extreme precipitation (Bell et al. 1995; den Hollander and Notenboom 1996). Elevated turbidity caused in part by rainfall may be responsible for a significant proportion of physician visits and hospitalizations for non-specific gastroenteritis in some urban areas (Aramini et al. 2000).

Projected changes in precipitation patterns in Ontario may affect waterborne enteric infection rates. Frequent mid-winter thaws and drier summers punctuated by heavy precipitation events may increase non-point source contamination of surface waters. Lower lake, stream and river levels due to evaporation may lead to higher concentrations of contaminants following heavy rain events of short duration. High turbidity or low flow levels may reduce the effectiveness of water treatment systems. Organism survival in water distribution systems is enhanced during warmer temperatures. Finally, lower Great Lakes levels may present drinking water quality issues to major uban areas in southern Ontario. All of these issues should enter into infrastructure planning now to help guard against a potential increase in waterborne illness in Ontario in the future.

b) Legionellosis

A range of clinical syndromes is associated with infection by *Legionella pneumophila* and other *Legionella* spp. The best recognized are Legionnaire's disease, a severe pneumonia, and Pontiac fever, an acute febrile illness without pneumonia. The organism is found in various bodies of water. The reservoir for *Legionella* seems to include protozoa that live in biofilms – those that occur in nature, as well as scale and films that form inside water storage containers and inside medical devices. This intracellular existence within protozoa and biofilms contributes to Legionella's resistance to the chlorination process (Atlas 1999). Outbreaks have been linked to stored treated water, including cooling tanks (e.g., Formica et al. 2000, Brown et al. 1999) and water distribution systems (Yu 2000). Factors that facilitate the growth of the bacteria include temperatures of 25-40°C, stagnation, scale and sediment. People with chronic lung disease, smokers, and the elderly are particularly susceptible to infection (Yu 2000). Most of the Canadian cases occur in Ontario, with 30-50 cases of Legionnaire's disease reported annually (HC-PPHB 2002a, electronic access).

Many cases of Legionnaire's disease are acquired in hospitals. The organism is often recovered from cooling towers, but cases are often attributed to the micro-aspiration of potable (tap) water rather than the cooling system (Sabria and Yu 2002). Other reports suggest that outbreaks are linked to atmospheric aerosols from institutional cooling towers (in particular, Brown et al. 1999).

A sustained warmer Ontario climate, particularly more frequent heatwaves, may increase the incidence of legionellosis. Longer hotter summers could prolong or enhance the growth of *Legionella* in wet cooling towers, increasing the potential for atmospheric aerosolization. Persons already vulnerable to the disease (the elderly and those with chronic lung disease) may need to be hospitalized more often or have increased need of various breathing apparatus such as oxygen nebulizers. In such a scenario, those most susceptible to the disease are brought into potentially contaminated environments. Enhanced surveillance for the organism in hospital environments, as well as around wet cooling towers of all types, will help protect against increases in the incidence of the disease. The impact of climate change on other sources of this disease is not known at present.

c) Leptospirosis

Leptospirosis is an infection caused by various serovars of the spirochete bacterium *Leptospira interrogans*. This zoonotic pathogen infects humans, dogs and other canids, swine, rodents, and various other small wild mammals, including raccoons (*Procyon lotor*). It also infects amphibians and reptiles. Stagnant water (canals, swamps, ponds) contaminated by infected animal urine is a common source of infection. Clinical syndromes include lymphocytic meningitis, hepatitis, nephritis, myocarditis and hemodynamic collapse (Tappero et al. 2000).

Leptospirosis is not reportable in Ontario or in Canada; hence, reliable surveillance information is not available. The disease is believed to be rare in humans in Ontario. Cases of leptospirosis in Ontario hunters have been linked to raccoons (Warshawsky et al. 2000). Various serovars cycle in domestic and wild animal reservoirs, including *L. grippotyphosa* (Prescott et al. 1999). The disease is prevalent in Ontario and Québec raccoon populations and in Ontario cattle and horses (Prescott et al. 1999, Mikaelian et al. 1997, Shapiro et al. 1999, Warshawski 1998;). A resurgence of leptospirosis (*pomona* and *grippotyphosa* serovars) in domestic dogs may prove to be both a sentinel for increased human risk from wildlife sources of the disease and a potential source itself (Hrinivitch and Prescott 1997, Carmichael 1999, Warshawsky et al. 2000, Kalin et al. 1999, Prescott et al. 1999).

Peak occurrences of leptospirosis have been associated with high precipitation levels (Vinetz et al. 1996). Outbreaks have been linked to recreational exposure to infected water (rafting, boating, swimming) (Morgan et al. 2002, Trubo 2001). Outbreaks linked to rodents have been associated with flooding in Central America. In Nicaragua, a case-control study of the 1995 epidemic found a 15-fold risk of the disease associated with walking through floodwaters (Trevejo et al.1998).

Changes in Ontario weather, in particular extreme rainfall events, may enhance the public health threat posed by leptospirosis. Physician awareness of the clinical syndrome and compulsory reporting of cases will enhance early detection of disease and accurate surveillance of future trends.

d) Cholera

Cholera is an acute and severe gastrointestinal infection with the bacterium *Vibrio cholerae*. It is nationally reportable in Canada. Symptoms include profuse diarrhea with dehydration and metabolic acidosis, vomiting, and collapse. The bacterium produces a toxin that causes fluid loss and intestinal cell death. The natural reservoirs of the bacteria are marine dinoflagellates, a type of plankton. The disease is generally transmitted by drinking water contaminated by infected sewage or by eating contaminated fish or shellfish (Seas and Gotuzzo 2000).

Cholera is highly infectious and prone to epidemic/pandemic distribution; it remains endemic in many affected areas. In cholera-endemic areas, the organism is generally maintained in an aquatic reservoir, many adults carry the disease asymptomatically, and the highest rate of illness occurs in children. In unfavourable environmental conditions, the *V. cholerae* becomes dormant. The organism may persist indefinitely, and although it cannot be cultured in this dormant state, it can re-activate once conditions become more favourable. Outbreaks are almost invariably associated with the warmest months (Seas and Gotuzzo 2000).

Cholera is endemic in Asia and Africa but exotic to Ontario. The most recent epidemic of *V. cholerae* O1 El Tor in South America in 1991 was the first after an absence of more than 50 years (Seas and Gotuzzo 2000). Of concern, a new reservoir of cholera has become established along the U.S. Gulf Coast (Blake et al. 1980). During 1995-2000, a total of 61 laboratory-confirmed cases of *Vibrio cholerae* O1 infection were reported in the United States. Of the 35 patients hospitalized, one died. Thirty-seven cases were related to travel outside the United States, and six were caused by consuming contaminated seafood from Gulf Coast waters. Antimicrobial resistance was present in one third of travel-related cases (Steinberg et al. 2001). In the year 2000, 56 countries reported a total of over 137,000 cases of cholera, resulting in close to 5,000 deaths. Eighty-seven percent of these occurred in Africa (WHO 2001). There were five cases of cholera reported in Canada in 2000; two were imported cases, and three were indigenous (WHO 2001, HC-PPHB 2002b, electronic access). Previously, the last reported Canadian cases were three imported cases in Alberta in 1988 (HC-PPHB 2002a, electronic access). In 2001, Ontario had three imported cases of cholera (Health Canada 2002a; WHO 2001).

Climate change may affect the worldwide distribution of cholera, altering the risk of the disease to Ontarians traveling abroad and to visitors entering Ontario from abroad. The potential for the disease to become established in Ontario is minimal, since Ontario's marine shores will remain too cold to support the vibrio. Yet a sustained warming of the Great Lakes and contamination by infected ballast water from ships could permit foci of the disease to establish. Sanitary regulations for marine traffic, ongoing surveillance of the global distribution of cholera, and ongoing stringent water quality regulations are already in place and should guard against chlolera taking hold in Ontario.

3.3. Other Communicable Diseases, Including Zoonoses

Many infectious diseases are transmitted by direct contact with an infected person or animal or by contact with infected material. Zoonoses are diseases that are transmissible between humans and animals (Schwabe

1994). Animal populations may act as a reservoir of disease, becoming a source of infection to people and sometimes other animals. Many of the diseases discussed in previous sections (vectorborne, foodborne, and waterborne) are zoonoses. Climate impacts on the ecology of wildlife and livestock reservoir populations may contribute to animal epidemics of zoonoses such as rabies. If these outbreaks occur near populated, agricultural, or recreational areas, the possibility of transmission to people would increase.

3.3.1. Rabies

In Ontario, rabies is a zoonosis of significant public health importance. This fatal viral infection of the central nervous system is mainly a disease of animals, but humans may become infected by a scratch or bite from an infected animal. If untreated, rabies is fatal in most cases (Bleck and Rupprecht 2000). Since 1925, 22 rabies deaths have been reported in Canada (HC-PPHB 2002a, electronic access). The most recent was a young boy in Québec who died in October 2000 presumably following a bite from a bat, either a silver-haired bat (*Lasionycteris noctivagans*) or eastern pipistrelle (*Pipistrellus subflavus*) (Turgeon et al. 2000, electronic access).

Rabies virus is a lyssavirus of the family Rhabdoviridae. It is unusual in that it can infect all warm-blooded animals and is found worldwide. Symptoms, characterized initially by flu-like symptoms, usually appear three to eight weeks after infection. As the illness progresses, neurological symptoms occur, such as excitement, anxiety, difficulty in swallowing, and pronounced hydrophobia. Delirium, convulsions, coma and finally death (usually due to respiratory paralysis) occur within six to seven days (Bleck and Rupprecht 2000).

The epidemiology of human rabies generally follows that of animal rabies. For example, in Africa where canine rabies is epidemic, human exposure is usually from dog bites. In places where dogs (and other companion animals) are immunized, human exposure is usually linked to wildlife (Bleck and Rupprecht 2000). Since 1924, there have been six cases in Ontario, the last occurring before 1970 (Varughese 2000, electronic access). Nonetheless, since 1958, post-exposure vaccination has been administered to more than 63,000 people in Ontario (Nunan et al. 2002).

Historically in Ontario, rabies outbreaks occurred primarily in dogs as isolated outbreaks. In the 1950s, a rabies epizootic of Arctic origin rapidly became enzootic in southern Ontario red foxes and striped skunks (*Mephitis mephitis*). From 1958 until 1991, Ontario reported more laboratory-confirmed rabid animals than any other state or province in North America. Most of those cases occurred in southern Ontario (MacInnes et al. 2001). Fortunately, advances in human vaccines and a highly successful oral vaccination program have dramatically reduced the rate of rabies in wildlife, resulting in a reduction in the incidence of post-exposure treatment of humans (Nunan et al. 2002). However, bat rabies appears to be on the rise in the United States (Hanlon and Rupprecht 1998) and remains a threat to Ontarians.

Human recreational activities and economic exploitation of wildlife resources have contributed to the reemergence of rabies as a major zoonosis in the United States (Hanlon and Rupprecht 1998). Climate-related alterations in the habitat, range and population dynamics (length of hibernation, breeding success, etc.) of reservoir hosts may affect the distribution of this disease in the province. The introduction of raccoon strain rabies from the southeast United States into eastern Ontario in 1999 has important implications. Although a raccoon vaccination program has dramatically reduced the spread of this strain of rabies in Ontario, the potential for human exposure remains (MacInnes et al. 2001). Raccoons are well adapted to urban/suburban environments, and eventually there may be increased risk to pets and humans associated with populations of urban raccoons. Climate change may also affect populations of other reservoirs of sylvatic rabies (foxes, skunks and bats), with consequences for public health.

3.3.2. Influenza

Influenza virus type A causes disease in humans and other animals, especially domesticated birds and swine. Classically, symptoms include fever, myalgia, loss of appetite, inflammation of the upper respiratory tract, sometimes progressing to bronchitis and pneumonia. People with compromised immune systems and those suffering from cardiovascular and respiratory diseases are at greater risk of severe illness and mortality. A strain of this virus was the historical source of pandemic influenza, a significant cause of human mortality early in the last century. Pandemic influenza was characterized by severe outbreaks occurring nearly simultaneously in all parts of the world, extremely rapid transmission, and high attack rates in all age groups, with high mortality especially in young healthy adults (Treanor 2000).

In 2000-2001, there were 654 laboratory-confirmed cases of influenza reported in Ontario, of which 342 were type A, mostly H1N1 strain (Macey et al. 2002, electronic access). Actual incidence is much higher with an estimated 10-25% of Canadians infected every winter (HC-PPHB 2002c, electronic access).

Every winter, epidemic influenza poses a major public health problem in North America, with increased hospitalizations and deaths from the disease (Treanor 2000). It is not strictly a seasonal illness (it occurs year-round in warmer climates) but exhibits a distinct winter seasonality in temperate climates, including in Ontario. The reasons for this seasonality are not well understood – some theories include the drying effect on the respiratory tract of outside cold air and heated indoor air, increased survival of the virus in dry warm air, and enhanced transmission amongst people confined indoors (HC-PPHB 2002c, electronic access).

Generally, influenza A viruses are adapted to a single host species of swine, horses, birds, or to humans. They are transmitted freely among individuals within that species, but on occasion they can cross the species barrier and infect a new host. The genes for all influenza viruses are contained within the influenza viruses maintained in wild aquatic birds, which serve as a primordial genetic reservoir (Swayne 2000). Influenza virus infections do not cause disease in wild aquatic birds.

Ecosystems changes that increase opportunities for contact between infected waterfowl and susceptible populations of domestic poultry or swine may increase the likelihood of viral infection. Pigs are susceptible to infection with both avian and mammalian influenza viruses, and they serve as intermediate hosts for the adaptation of avian influenza viruses for replication in mammals and as mixing vessels where re-assortment between avian and human viruses can be generated. It is believed that all human strains of the disease originally stem from wild birds and are passed to swine (often through poultry) before they become infective to humans and other mammals. Many new strains of influenza likely emerge in Asia because domestic and wild waterfowl, poultry, and swine are reared and sold in close proximity to one another (Webster 1998).

New strains of influenza arise in other parts of the world, including North America. An avian influenza virus (H4N6) was isolated in 1999 from an Ontario swine herd with clinical respiratory disease, the first documentation of a wholly avian influenza virus from pigs in North America (Karasin et al. 2000). Because influenza is also the most common virus found in Canadian wild duck populatios (Sharp et al. 1993), the source of the swine herd infection was linked to a nearby lake on which large numbers of waterfowl congregated each fall. The potential importance of the large North American swine herd in the epidemiology of influenza is not well understood. However, it is likely that climatic conditions affect wild duck population numbers and behaviour and thus environmental contamination with influenza virus. Clearly, surveillance of swine influenza in Ontario and worldwide is an important aspect of pandemic influenza surveillance (Olsen et al. 2000).

Influenza A viruses have also been identified as causing clinical disease in free-ranging marine mammals in Canada (Neilsen et al. 2001), and associated with epidemics of pneumonia in harbor seals on the New England coast (Geraci et al. 1982). The regular association of avian viruses with influenza outbreaks in seals suggests that transmission of these viruses to seals occurs often and may be an important link in the evolution of new mammalian strains (van Campen and Early 2001). Transmission of influenza virus from seals to humans has been reported (Webster et al. 1981). The role of climate in the epidemiology of influenza in sea mammals is not known, nor can inferences for human health be drawn at this time.

Despite the large number of influenza type A strains in birds and mammals, human infections seem to be limited to six genetic subtypes. Yet an outbreak of influenza in humans in Hong Kong in 1997 was due to a different subtype (H5N1) of avian origin. The disease was severe in 18 people diagnosed during this outbreak (Chan 2002). Although there were fears of the emergence of a new pandemic strain, the likelihood of person-to-person transmission was considered to be low. The cases were all confined to an island and may have been due to massive exposure to avian virus (Webster 1998).

It is not clear what role milder winters may have on the epidemiology of influenza in Ontario. Global climate change may foster epizootics of this disease in wild birds, Asian poultry and swine, or seals, which may lead to the emergence of new strains of influenza. Clearly, the emergence of new epidemic strains or the outbreak of a new global pandemic would have widespread impacts on human health in Canada and internationally. On the other hand, milder winters may weaken the severity of the annual influenza epidemic in Ontario.

3.3.3. Tuberculosis

Tuberculosis is caused by a group of bacteria, the *Mycobacterium tuberculosis* (MTB) complex, which includes *M. tuberculosis*, *M. bovis*, *M. africanum*, and others (Haas 2000). The avian strains, known as *M. avis* complex (MAC) are ubiquitous environmental pathogens and important agents of disease in poultry and swine. Animals are not considered important reservoirs of human disease, despite persistent fecal excretion of the organism. MAC is a threat to AIDS patients and others with dysfunctional immune systems, and those with chronic lung disease. No person-to-person transmission has been reported (Havlir and Ellner, 2000).

Tuberculosis can affect many organ systems, but most cases acquire pulmonary tuberculosis. The usual symptoms for pulmonary tuberculosis are persistent productive cough, fever, and weight loss. The disease is transmitted by inhaling infected aerosol, or more rarely, by ingesting contaminated milk or meat (Haas 2000).

Tuberculosis is an enormous public health problem, killing 2 million people each year. The global epidemic is growing and becoming more dangerous: In 1993, the World Health Organization declared tuberculosis a global emergency. Each year, approximately 8 million new infections occur. Areas of greatest incidence include southeast Asia, sub-Saharan Africa, and eastern Europe (Coisivi et al.1998, WHO 2000, electronic access). Nearly all new cases of disease occur in the developing world, with significant increases in Africa due in part to co-infection with HIV. About one third of the world's population is infected, with new cases increasing 3% per year. Of these, 10% will develop active tuberculosis. Few will receive treatment. An active case may infect as many as 15 people per year (WHO 2000, electronic access, Grondin 2002, electronic access).

M. tuberculosis is responsible for most cases in Canada. Although livestock and milk were historically important sources of human tuberculosis (*M. bovis*), improvements in surveillance, strict test and cull policies, and milk pasteurization have rid livestock of the disease in Canada. The recent decline in the incidence of tuberculosis in Canada appears to have levelled off, for reasons that are poorly understood. A similar plateau seems to have been reached in other countries with established market economies (WHO 2002e, electronic access). The demographics of the disease have also changed. The proportion of cases reported in Canadian-born non-Aboriginal people has declined from approximately 50% of cases in 1980 to 18% in 2000. In Aboriginal Canadians, the proportion of cases has varied slightly but has returned to the 1980 levels of 15% of all cases. Nonetheless, rates of tuberculosis continue to be much higher in the Aboriginal population than in the Canadian-born non-Aboriginal population (HC-PPHB 2002d, electronic access). Given the epidemic levels of the disease elsewhere, it is perhaps to be expected that cases in Canada are increasingly occurring in people born outside the country, 65% of all cases in 2000 (HC-PPHB 2002a, 2002d, electronic access). The rate of tuberculosis in Ontario in 2000 was 5.7 per 100,000 population (rate for Canada, 5.5), and only 15% of these were acute in Canadian-born patients (HC-PPHB 2002d, electronic access). Approximately 700 cases of tuberculosis are reported annually in Ontario (HC-PPHB 2002a, electronic access).

Climate change may affect rates of infection in Aboriginal populations, particularly in the North, as groups may be required to relocate due to changes in food availability and other ecological changes. Relocation and regrouping of populations enhances tuberculosis transmission. Climate change may also affect tuberculosis rates internationally. Drought, floods, and famine in developing countries where tuberculosis is epidemic result in social conditions that favour the transmission of the disease in humans and may displace refugees who then travel great distances and may either contract or transmit tuberculosis. Travelers to and from Canada should be aware of the potential public health issues associated with tuberculosis and submit to proper testing and treatment where necessary.

The zoonotic risk from sylvatic tuberculosis is not known. Livestock may become infected by sharing pasture with infected wildlife (Coisivi et al. 1998). The prevalence of tuberculosis among First Nations people is an on-going concern, as is the rising incidence of co-infection with HIV (Dankner et al. 1993, Coisivi et al. 1998). The potential impacts of climate change on tuberculosis in these groups and in the general population are not clear.

3.3.4. Brucellosis

Brucellosis is a disease caused by various species of the bacterium *Brucella*, including *B. aborta* (cattle), *B. melitensis* (sheep and goats), *B. suis* (pigs), and *B. canis* (dogs). In these species, it is a chronic disease and is transmissible to humans, in whom it also causes a chronic, recurring illness. People generally contract the disease by consuming un-pasteurized milk and milk products from infected cows and goats or by direct contact with infected animal tissues. Symptoms are notoriously variable and include recurring flu-like episodes with joint pain, depression, and weight loss. Brucellosis is treated with antibiotics (Young 2000). Like tuberculosis, brucellosis was formerly a production-limiting disease in livestock and a public health threat that was eliminated from Canadian livestock through test-and-cull programs.

Brucellosis is rare and declining in Canada, with between 3 and 17 cases reported annually since 1989. A few cases from Ontario are reported annually (HC-PPHB 2002a, electronic access). In the United States, the incidence of brucellosis peaked around 1975 and has declined to about 100 cases per year since 1990 (Young 2000).

Although data for Ontario are not available, endemic foci of both sylvatic brucellosis and tuberculosis remain in Canadian wild ungulates (Ferguson 1997, Meyer and Meagher 1995), and marine mammals (Forbes et al. 2000). Brucellosis can be transmitted from wildlife to livestock (Forbes and Tessaro 1996). Changing climatic conditions will affect habitat and food resource availability for wildlife and may change exposure patterns to livestock and humans. Other changes may include the introduction of southern species into the existing ranges of northern wildlife, including those of bison and caribou. Outbreaks of tuberculosis and brucellosis may occur in wild ungulates; thus, the disease could be spread to livestock and people, in particular hunters and people practicing a subsistence or traditional Aboriginal lifestyle. The use of more northern rangelands could lead to a resurgence of these diseases in livestock. Increased human recreational activities during milder summers may bring more people into contact with infected wildlife in the north, increasing the risk of sporadic human occurrences of disease.

3.3.5. Rodentborne Diseases

a) Plague

Plague is caused by infection with the bacterium *Yersinia pestis*, and is primarily an infectious disease of rodents and fleas. Humans become infected with plague when fleas leave a dying rodent host to feed on humans. The disease consists of three classical syndromes. The most common form is bubonic plague, which causes characteristic blackish lesions in lymph nodes near the flea bite. The glands become inflamed and may rupture and drain to the external skin surface. All forms of plague may progress to generalized spread in the bloodstream (septicemic plague) and have a fatality rate of 50-60% if untreated. Septicemia may result in pneumonia (pneumonic plague), the most contagious and fatal form of the illness (Butler 2000).

In the 14th and 15th centuries, pandemic plague decimated the European population. The disease persists in rats in South America, Africa, and Asia: Over 13,000 human cases (1,000 deaths) were reported worldwide between 1990 and 1995 (CDC, 2002j, electronic access). North American sylvatic plague is maintained in wild rodent populations in southern British Columbia and Alberta and in the western United States (Cheney 1998). Human cases of plague have not been reported in Canada since 1924, though fears that Norway rats (*Rattus norvegicus*) would spread plague from ground squirrels caused the launch of the Alberta Norway rat eradication program in 1950 (AAFRD 2002, electronic access).

Sylvatic plague occurs in rodent populations, primarily prairie dogs (*Cynomys ludovicianus*) in several areas in the United States and in Richardson's ground squirrels

(*Spermophilus richardsonii*) in two areas in southern British Columbia and Alberta (Leighton et al. 2001). Since fleas may carry the bacteria for months under suitable conditions of temperature and humidity, and rodent populations are affected by climate, climate change may alter the distribution of this disease (Gubler et al. 2001). Changes in land use patterns and in climate have been blamed for the recent increase in plague in the United States (Sarisky et al. 1994). Of the 362 Americans infected between 1944 to 1993, all occurred in Arizona, New Mexico, and Colorado until 1984, when cases suddenly spread to an additional 10 states north and east of the original three (CDC 1997a). Rapid urbanization in endemic areas has resulted in increasing numbers of persons living in or near active plague foci, and in increased human exposure. Endemic plague foci were strongly affected by the El Ninõ southern oscillation (ENSO) climate events from 1991-1993 and with increased rodent populations following the rains of 1993. Sylvatic plague activity usually peaks during or immediately after years with cooler temperatures and more rain than usual, conditions that favour rodent populations (Parmenter et al. 1999). An analysis of climate and human plague in the southwestern United States from 1960 to 1997 suggests that the risk of plague is closely linked to climate, most notably maximum daily summer temperature values and time-lagged (one and two year) amounts of late winter (February-March) precipitation (Enscore et al. 2002).

To date, sylvatic plague has not been detected in rodents in Ontario, which lies outside the current North American range of plague in North America (west of the Mississippi basin). The reasons for this distribution are not clear but may relate to the distribution of various species of ground squirrels. In endemic areas, plague infects many other species. It is not clear what factors would be required for the disease to spread rodent populations in regions that do not support ground squirrels. These large communal populations of rodents appear to be essential for the endemic establishment of the disease in modern North America. The impact of climate change on this apparent limitation is not known. Plague may pose a greater hazard to Ontarians traveling to endemic areas in future.

b) Hantavirus

Hantaviruses are Bunyaviridae and are parasites primarily of wild rodents and some insectivores. Unlike other bunyaviruses, hantaviruses are not arthropodborne. The group includes many viruses capable of causing hantavirus pulmonary syndrome (HPS), each with a preferred rodent host. The most important of these is sin nombre virus (SNV), carried by the deer mouse (*Peromyscus maniculatus*). New York virus, a variant of SNV, is carried by *P. leucopus* (white-footed mouse) in the northeastern United States and is of potential importance in Ontario. The white-footed mouse is also a reservoir of Lyme disease (Steere 2000). Symptoms of HPS include fever followed by acute pulmonary edema and shock. There is no specific treatment for HPS, and the disease is often fatal.

HPS was first recognized in May 1993 following the investigation of a cluster of unexplained deaths among Native Americans in the Four Corners area of the southwestern United States. Their illnesses were characterized by fever and acute pulmonary edema simulating acute respiratory distress syndrome. Subsequently, the SNV was isolated. HPS is now recognized as a pan-American zoonosis with an expanding clinical spectrum. Humans become infected by contact with infected rodents or their excretions. Infection is strongly associated with disturbing rodent urine, droppings or nests in closed-up spaces; once disturbed, viral particles become airborne and are inhaled. In North America, there is no evidence of secondary, person-to-person transmission. In South America, person-to-person transmission may be a factor in disease spread.

In Canada HPS is a nationally notifiable disease, and 36 cases have been reported between 1989 and 2001. Drebot et al. (2000) reviewed 32 cases of HPS occurring in Canada between 1989 and 1999. To date, cases of HPS seem to be confined to the western provinces (British Columbia, Alberta, Saskatchewan and Manitoba); however, the presence of infected mice in eastern Canada suggests that the potential for HPS exists across the country (Drebot et al. 2001, 2000). In Canada, the reporting of human cases follows a bi-modal spring and late fall distribution. The reason for this distribution is unknown, but a combination of human behaviour, rodent populations, and other ecological factors are proposed. In the United States, a total of 256 cases from 30 states were confirmed between 1993 and 2000. During 2000, a total of 41 (34 confirmed) cases of HPS were reported from 10 states. Seven of the infected people died (CDC 2000b).

Increased small mammal populations may foster animal epidemics of HPS. For example, the HPS epidemic in the southwest U.S. was thought to be due to an upsurge in rodent populations related to climate and ecological conditions (Glass et al. 2000, Wenzel 1994). Six years of drought, followed by extremely heavy spring rains in 1993, resulted in a 10-fold increase in the local population of deer mice, which are reservoirs for the hantavirus (Stone 1993). Another outbreak was associated with the 1997-1998 ENSO events (Hjelle and Glass 2000).

In Canada, Drebot et al. (2000) noted that the number of cases per year fluctuated from a high of eight in 1994 to two in 1999. One case was reported in 2000 and three cases in 2001 (Health Canada 2002a). The highest number of cases in Canada coincided with the warmest year on record and the 1997-1998 ENSO events. Yearly fluctuations in cases may reflect that population densities and breeding capacities of infected rodents increase in mild winters (Mills et al. 1999), and that the increased prevalence of mice may lead to increased opportunities for human exposure to rodents and their excreta.

Like that of other rodentborne diseases (e.g., plague, Lyme disease), the risk of HPS may increase where climate change creates conditions favorable to rodent populations. Ontarians traveling to endemic areas may face greater hazards from hantaviruses. Climate change may alter rodent ecology in Ontario and may change the likelihood of human exposure.

3.3.6. Parasitic Infestations

a) Hydatid Disease (Echinococcosis)

Two of the four species of *Echinococcus*, *E. granulosus* and *E. multilocularis* are recognized public health issues in Ontario. The adult tapeworm lives in the intestines of adult canids (wild and domestic) and sheds proglottids containing eggs, which are passed in feces. The eggs are then ingested by herbivores. Subsequently, the larvae encyst within the liver or other organs of the herbivore. The cycle is closed with the predation of the herbivore by the canine predator, which then ingests of mature cysts found in the flesh of the herbivore (Roberts and Gemmel 1994).

Humans are accidental hosts of *Echinococcus* species. Cystic hydatid disease is usually a mild clinical syndrome caused by the larval form of *E. granulosus*. Alveolar hydatid disease is a more serious clinical problem caused by *E. multilocularis* (Roberts and Gemmel 1994). Infestation results in small metastases throughout many organs and is often fatal. Hydatid disease is borne by blowflies (Lawson and Gemmel 1985), but is also foodborne; it has the greatest impact on the rural poor. *E. granulosus* has worldwide distribution. *E. multilocularis* is confined to northern hemisphere and cycles between the arctic fox (*Alopex lagopus*) and red fox and rodents of the genera *Microtus*, *Lemmus* and *Clethrionomy*.

Environmental factors determine the lifespan and infectivity of the parasite. *E. granulosus*, for example, is not found in hot, dry parts of Australia (Gemmel 1990). *E. multilocularis* is endemic in mountainous areas of central Europe, where the prevalence in foxes reaches 40% and where low temperatures preserve the eggs sufficiently long enough to allow infection of rodents (Delattre et al. 1991).

In the Canadian northwest, hydatid disease due to *E. granulosus* has typically been asymptomatic and usually benign, unlike the more aggressive pastoral form of the disease. Clinical cases have occurred in Aboriginal people, in particular among children. Domestic dogs fed entrails of wild ungulates are often the source of the disease (Lamy et al. 1993, Unruh et al. 1973). A serological study of 115 fox trappers in South Dakota in 1990-1991 found no serological evidence of *E. multilocularis* despite a very high prevalence in area foxes (Hildreth et al. 2000). However, serological testing may not be very sensitive (Lamy et al. 1993). The native Canadian disease usually resolves spontaneously, does not cause anaphylaxis, and does not implant daughter cysts if spilled. Surgical treatment should be avoided except for complications such as secondary bacterial infection (Lamy et al. 1993).

Environmental contamination by feces from infested canines is the principal source of human infestation. The eggs of this parasite must be ingested; lapses in hygiene and the incomplete washing of contaminated crops (berries, herbs and greens, etc.) likely play a role in human disease (Hildreth et al. 1991). Climate change might lead to changes in habitat that then alter the range and habits of wild canids or predation behaviour by domesticated dogs and cats. As a result the level of contamination in the environment could change, leading to focal increases in disease incidence particularly in Aboriginal peoples.

b) Visceral Larva Migrans

Visceral larva migrans (VLM) is a disease of humans (usually children) caused by reaction to normal encysting behaviour of larvae from animal ascarid parasites. The most common cause of VLM are larvae from the cat and dog roundworms (*Toxocara cati* and *T. canis*). Less commonly, the raccoon roundworm (*Baylisascaris procyonis*) or various canine *Dirofilaria* can cause VLM (Chitkara and Sarinas 1997, Rowley et al. 2000).

Clinical manifestations of VLM are the result of allergic and inflammatory responses of the host and include airway reactivity, acute pneumonia, and persistent eosinophilia. In a review of American cases, Glickman et al. (1979) found that the typical patient with serologically confirmed toxocaral VLM was about five years of age and resided in the southern half of the United States. Clinical findings were likely to include leucocytosis, eosinophilia, an increased anti-A or anti-B isohaemagglutinin titre, and an elevated serum IgG level. Pica, as ascertained by a physician questionnaire, was not a consistent finding, and both sexes were nearly equally represented.

VLM is a self-limited disease, and specific treatment is rarely necessary. In acute cases, a short course of steroids reduces morbidity and mortality, but preventive measures are more important in curbing *Toxocara* infection (Chitkara and Sarinas 1997). Severe illness may occur, for example eosinophilic meningoencephalitis, retinitis, and a protracted encephalopathy with severe residual deficits from *Baylisascaris* VLM (Rowley et al. 2000). Chronic sequelae of VLM have been suggested by Nelson et al. (1996); however, they found that rather than being a consequence of VLM for disadvantaged children, lower initial intelligence and less advantageous child rearing are risk factors for *T. canis* exposure. Infestation with the adult stage of *Toxocara* may also occur, as reported by Eberhard and Alfano (1998). The affected children (aged 20 months to seven years) showed no symptoms beyond rectal expulsion or vomiting of worms. No symptoms or serological evidence of VLM occurred.

The potential impact of climate change on VLM in Ontario is not known. Since most cases are due to exposure to dog or cat feces, no great change is expected in the risk of this disease in Ontario children in future. However, changes in raccoon population dynamics as a result of climate change may alter the environmental contamination levels of *Baylisascaris* larvae, causing public health concerns.

4. Conclusion

Global climate change will affect the lives of people in Ontario in innumerable and complex ways. Some health impacts could be very significant, some more subtle. A sustained change in climate in Ontario will affect health directly (examples: heat stress and respiratory problems during heat waves, elevated rates of skin cancer or cataract associated with increased UV radiation, fewer deaths due to cold). More insidious but perhaps more significant are indirect health impacts that may result from changes in the ecology of infectious disease reservoirs, vectors and transmission dynamics.

Ontario contains large and important populations of domestic animals and wildlife, including fish and birds. The dynamics of infection in animal populations are likely to be affected by climate change, given the generally more intimate connection between animals and their environment. Some of these infections are zoonoses and are of potential direct public health significance. However, even infections that do not directly affect humans may alter animal population dynamics in a way that may cause other diseases to emerge, and that may, in turn, affect human health. The ecology of many infectious diseases is already changing in Ontario (indeed, globally), with bacterial acquisition of new virulence factors and drug resistance, the emergence of new diseases (e.g., West Nile virus), disseminated patterns of exposure through commercial food distribution systems, and the illnesses following devastating waterborne outbreaks of *E. coli* O157:H7. We know that socioeconomic and demographic factors are important health determinants and that climate change may have social and economic impacts that could change disease patterns in Ontario. The connections between socioeconomic human behaviour systems and disease ecology are overwhelmingly complex. How will climate change alter these dynamics?

Although precise predictions of the impact of climate change on the already highly complex ecology of infectious diseases are not feasible, it is possible to make some informed projections based on our understanding of how climate affects infectious diseases now, and by including a wider perspective that includes socioeconomic systems and responses and uncertainty. In practical terms, this means continuing to learn how infectious diseases behave in the environment (including human environments), how climate and weather affect health today, and how, where and for whom a changing climate may alter all this in the future.

As our understanding grows, so will we be better able to adapt to the health impacts of climate change in Ontario. While efforts are applied to reducing greenhouse gas emissions and other climate change mitigation strategies, simultaneous attention must be given to adaptation planning. Even under utopian scenarios of rapid and global cessation of greenhouse gas emissions, a certain degree of global warming is inevitable (IPCC 2001). Hence, adaptation plans are needed now, and such plans must be responsive to new information as it becomes available. This report forms some of the basis of the information needed for adaptation planning.

Valuable knowledge on the health impacts of climate has been acquired through a number of recent public health crises. Since the mid-1990s, the rising health impacts of severe heatwaves in southern Ontario have lead to health alert systems and heat emergency response planning for Toronto and, increasingly, other cities. The *E.coli* O157:H7 tragedy in Walkerton in May 2000 exposed the vulnerability of drinking water to contamination following extreme rainfall and provoked a complete reassessment of drinking water safety in the province. It resulted in not only more stringent water quality standards but also more stringent management, training, and reporting systems, and a mandated overhaul of water treatment infrastructure. Measures for the protection of source waters are being prepared. Finally, the emergence and spread of West Nile virus in Ontario since 2001 has revealed how quickly a vectorborne disease can be distributed in a new but suitable environment. Researchers are investigating the role of climate in the WNV epidemic, with an eye on climate change and the introduction of still other exotic vectorborne diseases.

How should planners set priorities among so many different public health issues? Given our current understanding of climate and disease in Ontario, the authors suggest that public health climate change adaptation planning for Ontario should include consideration of the following issues as they occur in Ontario, in neighbouring regions, and further abroad:

- Minimizing the health impacts of more severe heatwaves and smog, particularly in urban areas
- Protecting vulnerable populations in Ontario (Aboriginal people, northern communities, urban poor, the elderly, immigrants) through targetted surveillance and health promotion and protection programs
- Enhancing surveillance of mosquitoborne and tickborne diseases, particularly arboviruses and illnesses transmitted by the black-legged tick
- Protecting people from waterborne infections, for example, through developing livestock management practices that reduce environmental contamination with human pathogens and protecting source water from agricultural runoff, sewage and industrial waste (of particular concern are protozoa and bacteria resistant to antimicrobials)
- Surveying zoonotic diseases in wildlife, including rodentborne diseases, as animal populations respond to a changing climate
- Considering factors from farm to fork that might contribute to increased foodborne disease in Ontario; antimicrobial resistance is of particular concern in the emergence of foodborne diseases
- Considering changes in the epidemiology of exotic diseases and the implications for travelers

This synopsis of human diseases potentially affected by climate change in Ontario is intended for public health researchers and policymakers. While it does not attempt to project how climate change will affect specific diseases, it has outlined how various diseases might be vulnerable to climate change. There are enormous knowledge gaps and considerable uncertainty exists about knowledge presently available. Nonetheless, initial steps toward adaptation planning must be made now.

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4.3. Personal Communication

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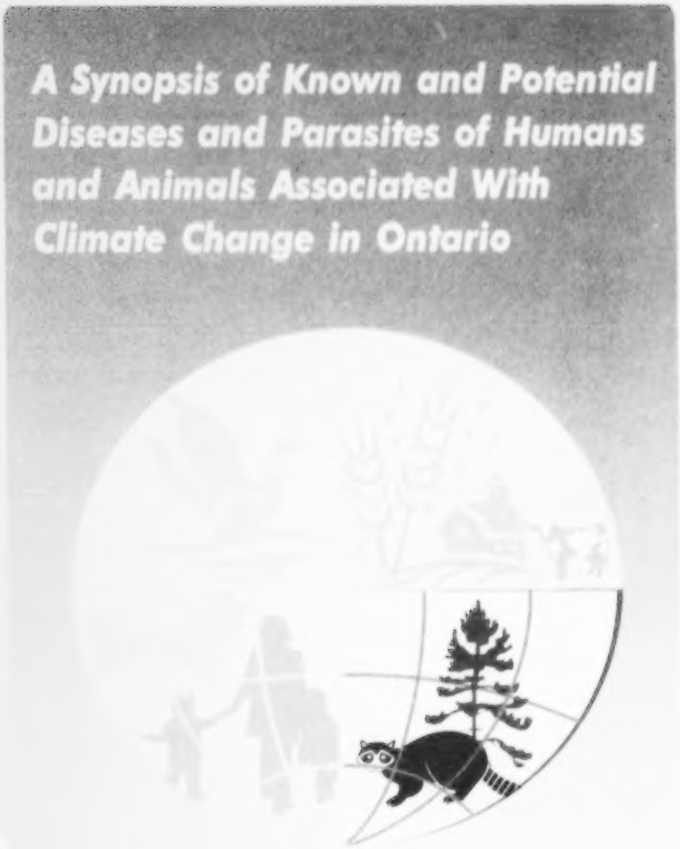

A Synopsis of Known and Potential Diseases and Parasites of Humans and Animals Associated With Climate Change in Ontario

Part C.

Diseases and Parasites of Animals Associated With Climate Change in Ontario

1. Introduction

Diseases in animals are important for the animals that suffer from them and for the people who raise them, eat them, and/or share environmental spaces with them. Many of the agents that infect animals can be transmitted to people (zoonoses such as the rabies virus, BSE, *E. coli* 0157:H7 and a variety of intestinal parasites). Other agents may have sufficient impact on agricultural animals that the livelihoods of farmers and rural communities may be negatively impacted, often severely. The recent epidemics of foot and mouth disease in the United Kingdom and classical swine fever in the Netherlands are extreme cases in point.

We know, furthermore, that many animal diseases in both domestic and wild animals occur seasonally, that animal behaviours can vary in response to weather variables, and that these changes influence patterns of transmission among animals. Indeed, farmers who raise livestock in Ontario - as in most of the world - have always had to manage their animals to adapt to diurnal, seasonal weather variability. They pasture, house, and feed their animals differently throughout the year and manage reproductive cycles to take advantage of seasonal changes. Beef cattle and sheep, for instance, have traditionally been managed to give birth in the spring, when pasturage becomes available.

We also know that both disease-causing agents and their vectors are strongly affected by winter. Many adult insect vectors and the agents they carry are killed by low winter temperatures, so that cycles of disease transmission are at least interrupted and need to be re-started in spring.

Given the importance of animal diseases and their responses to weather variables, it may seem strange that the public discourse on the effects of climate change rarely incorporates considerations of the health effects on animals. This may be because farmers have learned to protect their animals from the worst ravages of weather and seasonal changes, often through fairly large investments in feeds, housing, and energy use. Urban dwellers have been sheltered from both livestock and wildlife diseases by a perceived urban "protective" effect (what you don't know can't hurt you) or have adapted through changes in clothing, behaviour and pesticide use, often without being fully aware of the reasons for these changes.

Recently there have been high-profile disease outbreaks, which have often been zoonotic in nature (e.g., cryptosporidiosis and *E.coli* 0157:H7-related diseases). These outbreaks, which were associated with unusual precipitation patterns, have begun to change public perception of the importance of climate change and its possible disease consequences and the link that these outbreaks have to infections in animals. Understandably, the public would like to see policies in place as soon as possible to prevent or mitigate climate-associated diseases in general and animal-associated diseases in particular. Diseases associated with livestock infections appear to be most vocally targeted for prevention (e.g., in terms of factory farms), while diseases associated with wildlife, such as Lyme disease or West Nile virus-associated disease, tend to elicit calls for greater medical attention rather than pre-emptive government policies.

In any case, before we can design effective policies, we need to address some important questions: 1) Which infections and infestations of animals are most vulnerable to influences from climate change, and at which points in the maintenance cycle of the agent are weather effects most critical? 2) What kinds of adaptations are livestock owners, other animal owners, outdoor enthusiasts and public health officials now using to prevent or mitigate the effects of these infections and infestations? 3) To what extent are these adaptations likely to be effective under projected scenarios of climate change, and what other adaptations might be required?

This report addresses the first of these questions, particularly for Ontario, based on a thorough review of the scientific and technical literature and consultations with experts on the relevant topics. In each case, we have laid out the basic disease and the agent associated with it, its current status in Ontario, and how this status might be - or already is - affected by climate changes.

In general, climate affects the health status of animals either directly, i.e., through extremely high or low temperatures or through catastrophic natural events, or, more often, indirectly, by influencing the agents, vectors and ecosystems within which animals live (see Part A of this report, tables A.6-A.9).

1.1. Direct Effects

Climate change in Ontario is likely to bring with it more extreme weather events such as droughts, floods and heat waves (IPCC 2001, electronic access). These events can result in immediate physical harm to wild animals, and, because adaptive strategies now used by animal owners may be inadequate, to animals in home and farm environments. Potential direct effects of climate change on animals include direct mortality from extreme events such as tornadoes and ice storms, reduced performance in the case of livestock (e.g., reduced feed efficiency, growth rate, milk and wool production, reproduction), and altered disease susceptibility directly associated with thermal stress (linked to air temperature, humidity, wind speed and thermal radiation) (IPCC 2001, electronic access).

Pets are likely to suffer heat stress similarly to their owners. Since dogs (*Canis domesticus*) cannot perspire and often share closed urban environments with people (for instance, in cars) they are particularly susceptible to heat stress. With an increase in the frequency and intensity of heat waves, we can expect to have an increase in reports of mortality related to heat stress in these animals. As with people, animals suffering from cardiovascular or respiratory diseases are the most vulnerable.

1.2. Indirect Effects

Indirect effects of climate change include climatic influences on quantity and quality of feedstuffs (pastures, forages, and grain) and the severity and distribution of livestock diseases and parasites (IPCC 2001, electronic access). Furthermore, climate change and the resulting changes in complex biological systems will influence the incidence and distribution of certain infectious diseases, especially those that are vectorborne, such as the viral encephalitides. Increased temperatures and altered rainfall are likely to affect the range and biological behavior of vectors and of intermediate, amplifying or reservoir hosts, as well as the viability and maturation rates of infective agents such as bacteria in the environment. Indirect effects of short-term extreme events can include power outages that can affect intensively managed livestock as was seen after the 1998 ice storm in eastern Canada (Kerry et al. 1999).

The responses of disease agents to specific climate changes are difficult to predict. Organisms and ecosystems respond to changes in climate in a context of other landscape and socially induced changes and are often non-linear and defy exact prediction (Sutherst 2001, Kay et al. 1999). As an example, small changes in temperatures around developmental, survival or behavioral thresholds can result in sudden and sharp responses in the population. Similarly, temperature changes within the range of rapidly changing rates of growth or stress accumulation are likely to result in disproportionate responses by populations. Multiple, differential population changes may be reflected in changing biotic-a-biotic interactions, that is, a change in the organization of the ecosystem itself. This change in the organization can then feed back to affect populations in unexpected ways.

Because of the inherent uncertainty of these systemically mediated effects, and because these effects are likely to far outweigh direct effects, they pose a particular challenge to scientific understanding and policy response. We shall return to this challenge in part D of the report.

Some animal diseases are restricted to wildlife and others to animals kept by people for food or companionship. However, many disease agents - indeed probably most of those of concern under conditions of climate change - are *protean*, meaning they can infect multiple species, including people. Following is a description of these diseases.

2. Diseases Important for Multiple Species and Zoonoses

2.1. Diseases Transmitted Directly Between Animals/Species

A variety of disease agents that affect multiple species and are transmitted directly between animals (that is, they do not require an insect vector or a period of development external to the animals) may be influenced by climate change. Climate may favour the reproductive success and population densities of the affected species, and hence increase the probability of adequate contact within and between species. In addition, climate variability may lead to a change in the range over which wildlife can live, as well as expand the limits of agricultural activities, which may result in contact between species that do not normally interact in that area. Following are several examples of these kinds of diseases that are relevant to Ontario.

2.1.1. Influenza

Generally, influenza A viruses are adapted to a single host species (humans, swine, horses, birds, etc.) and are transmitted freely among individuals within the same species, but these viruses can cross the species barrier and infect a new host. The genes for all influenza viruses are contained within the influenza viruses maintained in wild aquatic birds, which serve as a primordial genetic reservoir (Swayne 2000). There are two major genetic lineages of avian influenza viruses, related to migratory paths of aquatic birds in Eurasia and North America. These enteric influenza virus infections in wild aquatic birds are not believed to cause disease.

Alterations of ecosystems that increase opportunities for contact between susceptible populations of wild birds and domestic poultry or swine may increase the likelihood of viral transfer and infection. In poultry, influenza infections can cause clinical respiratory disease or a drop in egg production. A few specific avian influenza virus strains have caused severe disseminated infections with systemic disease and high death losses. Many factors, such as geographic restriction, intermixing of species, age and density of birds, weather, and temperature impact the ability of the avian influenza virus to move within and between host species and affect the overall incidence of infections.

In March, April and May 2002, nearly 5 million birds from approximately 200 Virginia poultry farms were destroyed to control an outbreak of avian influenza, at a cost to farmers of more than US $140 million. Mexico and Russia banned the import of poultry and eggs from Virginia and other states with confirmed avian influenza cases. It is believed that cool, wet conditions in March and April 2002 encouraged the spread of the disease and that subsequent hot and dry weather slowed its progression (ProMed mail 2002, electronic access).

Mildly pathogenic avian influenza viruses have been transferred from free-flying birds (primarily mallards [*Anas platyrhynchos*]) to range turkeys (*Meleagris gallopavo*) reared outdoors (in Minnesota), during annual wild-bird migrations (Halvorson et al. 1985). In the fall of 1999, the Animal Health Laboratory isolated an avian influenza virus typed as H4N6 from a swine herd with clinical respiratory disease (Karasin et al. 2000). This is the first documentation of a wholly avian influenza virus from pigs (*Sus domesticus*) in North America. This virus is the most common virus found in the Canadian wild duck population. The swine herd was housed near a lake, on which large numbers of waterfowl congregate each fall, and water used on the farm was sometimes drawn from the lake. Pigs are susceptible to infection with both avian and mammalian influenza viruses, and they serve as intermediate hosts for the adaptation of avian influenza viruses for replication in mammals and as mixing vessels where reassortment between avian and human viruses can be generated.

Influenza A viruses have been identified as causing clinical disease in free-ranging marine mammals, associated with epidemics of pneumonia in harbor seals (*Phoca vitulina*) on the New England coast (Geraci et al. 1982). The regular association of avian viruses with influenza outbreaks in seals suggests that transmission of these viruses to seals occurs frequently and may be an important link in the evolution of new mammalian strains (van Campen and Early 2001). Transmission of influenza virus from seals to humans has been reported (Webster et al. 1981).

The first outbreak of avian influenza A(H5N1) virus in humans occurred in Hong Kong in 1997. Infection was confirmed in 18 individuals, six of whom died. Infections were acquired by humans directly from chickens (*Gallus gallus domesticus*), without the involvement of an intermediate host. The outbreak was halted by a territory-wide slaughter of more than 1.5 million chickens at the end of December 1997 (Chan 2002). Weather or climatic links in such outbreaks have not been studied, but high temperatures, leading to increased shedding of viruses from birds, changes in airborne transmission rates and changes in how animals are managed, may all interact to affect occurrence.

Historically, we have tended to view the emergence of new influenza viral strains as a peculiar characteristic of South Asian agricultural systems and climates. However, as some of the examples above suggest, climate changes that alter behaviour and infection rates in populations of wild birds (for instance, changes in overwintering patterns, as we have already seen in Canada geese (*Branta canadensis*), and in migration routes), combined with changes in animal management (e.g., increased ventilation or outdoor rearing because of heat waves), may increase the likelihood of viral transfer and infection in domestic animals in Ontario. Similarly, changes in ocean coastal temperature could influence the occurrence of influenza in seals.

2.1.2. Rabies

Rabies is a disease of neurological tissues in mammals, including people. It is caused by a virus (Lyssavirus subgroup within Family Rhabdoviridae) and is usually transmitted by direct contact such as bites. Once clinical signs (usually abnormal behavioural patterns) are evident, the the victim always dies. Both vaccination and post-exposure treatment appear to be effective. Fox rabies entered Ontario from the Arctic in the early 1950s. From 1958-91, with the exception of 1960-62, Ontario reported more cases of rabies than any other state or province in Canada and the United States. The numbers peaked in the late 1980s at over 3000 cases annually. Most of these cases were in wildlife, especially foxes (Vulpes vulpes), and occurred in the densely settled southern 10% of the province. Rabid wild animals serve as reservoirs of infection for livestock and companion animals, which in turn increases the risk of human exposure. Since the late 1980s, an oral vaccination baiting program of foxes carried out by the Ontario Ministry of Natural Resources has all but eliminated fox rabies, as well as related cases in skunks and other species, including livestock, from the province. Current concerns are related to an epidemic of a raccoon (Procyon lotor) strain of rabies that has moved up the eastern United States and has, since 1999, made a few brief forays across the border. Concerted and quick responses have quickly contained cross-border incursions up to this writing (Figure C.1). However, increasing density and dynamic interactions in the raccoon population related to urbanization and temperature increases may render this control [illegible]. The dynamics of bat rabies is less well understood, but the disease appears to be widespread; the last two Canadian cases of human rabies, in Alberta and Quebec, have been from bat exposures.

Rabies, at first glance, is not an obvious candidate for being influenced by climate. Clearly changes in landscape structure (urbanization, increased habitat for raccoons and other small mammals) and intrinsic strains of infected

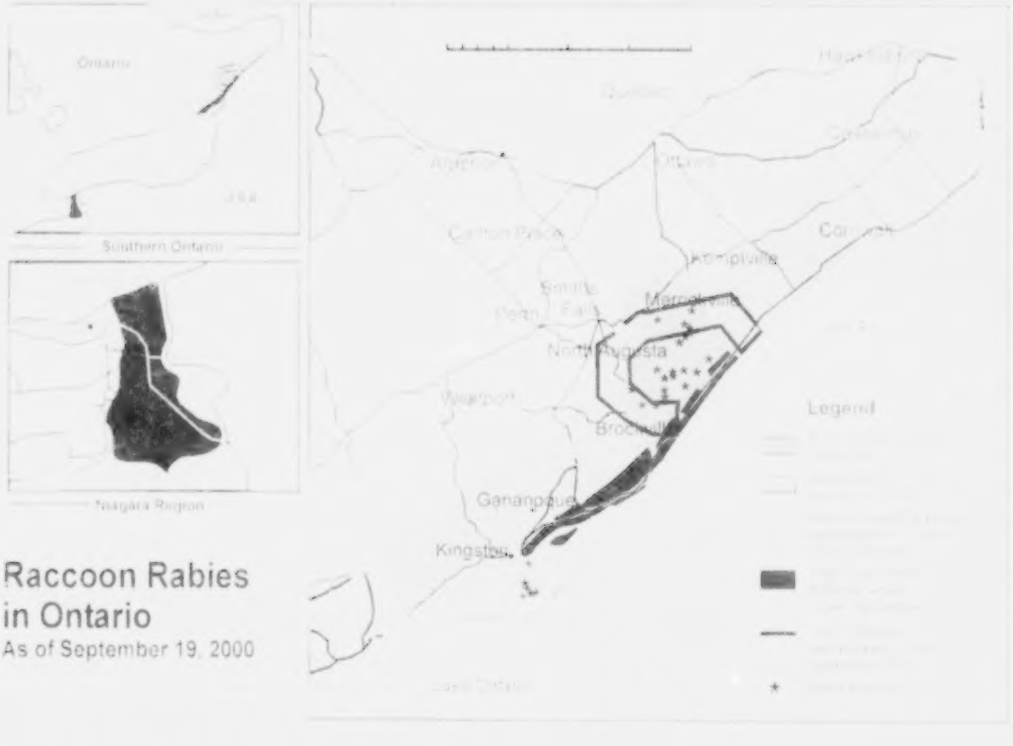
Ontario
U.S.A.
Southern Ontario
Niagara Region
Raccoon Rabies
in Ontario
As of September 19, 2000
Quebec
Ottawa
Arnprior
Carleton Place
Cornwall
Kemptville
Smiths Falls
Perth
Merrickville
North Augusta
Westport
Brockville
Gananoque
Kingston
Legend
Lake Ontario

Figure C.1 Raccoon rabies in Ontario, 2000 [illegible] Ontario Ministry of Natural Resources, [illegible]

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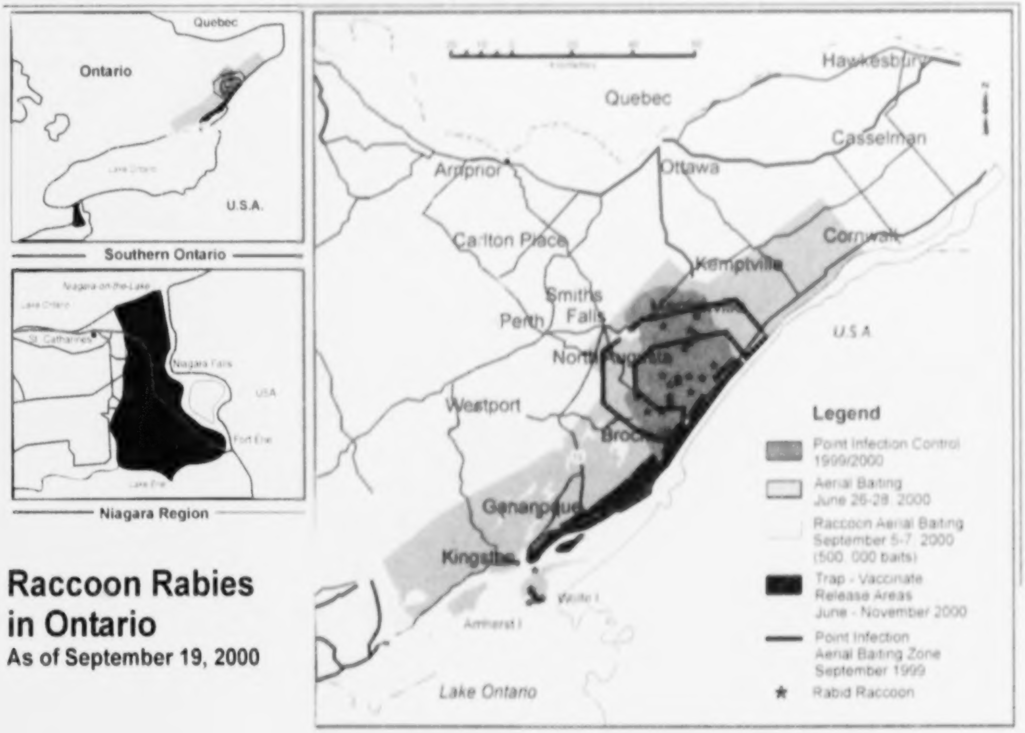
Quebec
Ontario
Lake Ontario
U.S.A.
Southern Ontario
Niagara-on-the-Lake
Lake Ontario
St. Catharines
Niagara Falls
USA
Fort Erie
Lake Erie
Niagara Region
Raccoon Rabies in Ontario
As of September 19, 2000
Hawkesbury
Quebec
Casselman
Arnprior
Ottawa
Carleton Place
Cornwall
Kemptville
Smiths Falls
Perth
North Augusta
U.S.A
Westport
Brockville
Gananoque
Kingston
Wolfe I.
Amherst I.
Lake Ontario
Legend
Point Infection Control 1999/2000
Aerial Baiting June 26-28, 2000
Raccoon Aerial Baiting September 5-7, 2000 (500, 000 baits)
Trap - Vaccinate Release Areas June - November 2000
Point Infection Aerial Baiting Zone September 1999
Rabid Raccoon

Figure C.1. *Raccoon rabies in Ontario, 2000 (courtesy of the Ontario Ministry of Natural Resources, Peterborough).*

populations are major driving forces of the epidemiology of the disease. However, temporal variations should make us cautious. Historically, rabies occurrence in foxes in Ontario has followed both seasonal and 3-4 year cycles (MacInnes et al. 2001). The seasonal cycles are believed to have been due to dispersal, territorial competition and mating activities; the longer-term cycles are thought to reflect recovery of populations from the devastation of rabies dieoffs. Projected warmer winters could be expected to alter hibernation and perhaps breeding, seasonal density and dispersal patterns of these animals, and hence alter the transmission and epidemiology of rabies in Ontario. Higher animal density may make containment of raccoon rabies more difficult.

2.1.3. Canine Distemper

Canine distemper is an acute, often lethal, infectious respiratory and sometimes gastrointestinal disease of Canidae (e.g., wolves, foxes, coyotes, dogs), Procyonidae (e.g., raccoons), Ursidae (e.g., bears), Mustelidae (e.g. skunks, otters, weasels) and Hyenidae (hyena family). It is caused by a morbillivirus and is characterized by fever, dullness, loss of appetite, and a discharge from the eyes and nose. This viral disease is spread most often when animals come in contact with the bodily secretions of other infected animals, but airborne spread is also possible. It is largely controlled in pets through vaccination, but outbreaks occur regularly in wildlife in Ontario, particularly raccoons. Half of all raccoons in the Greater Toronto area may have been exposed to the virus; however, disease varies widely from area to area and year to year (Schubert et al. 1998). While it does not appear to be a major constraint on raccoon populations themselves, the disease may be confused with rabies and poses a threat to recovery programs for endangered species such as the black-footed ferret (*Mustela nigripes*) at the Toronto Zoo. Vaccination programs for distemper in raccoons in the Toronto area are currently effective in reducing the incidence of the disease. Any effects of climate change on distemper will likely be mediated similarly to rabies, that is, milder winters may affect reproduction and population dynamics.

2.1.4. Tuberculosis and Brucellosis

Tuberculosis (TB) and brucellosis are zoonoses that in Canada have been traditionally associated with ruminant livestock species and have been the target of major, expensive, and largely successful, national eradication programs. For this reason, and because both remain problematically enzootic in certain wildlife populations in Canada, they are grouped together here.

TB is a bacterial disease that causes tubercles to form in various organs of a variety of mammals. Infected animals may have no symptoms; however some animals may become emaciated and develop chronic respiratory problems. *Mycobacterium bovis* and *M. avium* are the two most common causative agents in animals. *M. tuberculosis* and *M. africanum* are the main causative agents in people, where pneumonia has been the usual clinical manifestation. All can cross species barriers (Radostits et al. 2000, Acha and Szfres 2001).

In Canada, *M. bovis* infection has been largely eradicated from domestic animals. Infection still occurs in a few pockets of free-living and captive deer, elk (*Cervus elaphus*), and bison (*Bison bison*) in Alberta and Manitoba (Koller 2001). These wild populations serve as potential reservoirs of infection and as targets for spread from domestic animals (Clifton-Hadley et al. 2001). An infected beef bull (*Bovis domesticus*) was detected in Manitoba in 2001 (CFIA 2001a, electronic access); in June of 2002, an infected herd of dairy cattle was detected in Ontario, near Peterborough. The source of this infection was unknown at the time of this writing. A focus of bovine TB exists in free-ranging white-tailed deer (*Odocoileus virginianus*) in northern Michigan, in similar climatic and landscape conditions to those found in Ontario. Circumstances of high density, such as concentration of deer in winter yards as a result of supplemental feeding, may contribute to the persistence of TB in free-ranging deer. *M. bovis* has also been isolated from other wildlife in the area, including coyotes (*Canis latrans*), raccoons, a bear (*Ursus americanus*), and a fox. Once a wildlife reservoir has been established, it is very difficult to eliminate, and the presence of this wildlife reservoir may prevent the United States from achieving the goal of eradication of TB in animals (Whipple and Palmer 2000). The degree of risk that these infected deer pose to other wild and farmed animals is unknown; it will vary depending on the degree of contact with infected animals, population density of the reservoir and prevalence of infection.

Brucellosis is another zoonosis that has been the target for national eradication programs in Canada's livestock. It has been described under a variety of names, such as undulant fever, Malta fever, and Bang's disease. Although brucellosis can attack other animals, its main threat is to cattle, bison, and swine. Infection in animals causes decreased milk production, weight loss, loss of young, infertility, and lameness. The disease is caused by a group of bacteria of the genus *Brucella. B. abortus* principally affects cattle and bison, and *B. suis* principally affects swine and reindeer (*Sorex cinereus*) but also cattle and bison (APHIS 2002, electronic access). Brucellosis also affects goats (*Capra hircus*), sheep (*Ovis aries*), caribou (*Rangifer tarandus*), elk, dogs, and coyotes (HC-PPHB 2001a, electronic access). It has been acquired by Inuit people in Canada's far north, probably through ingesting raw caribou meat (Chan et al. 1989. Brucellosis is commonly transmitted by direct contact with infected animals or with an environment that has been contaminated with discharges from infected animals.

Canadian livestock herds have been considered free of brucellosis since the late 1980s (CFIA 2001a, electronic access). However, in Canada, *B. abortus* occurs in bison in Wood Buffalo National Park in Alberta (Forbes and Tessaro 1996) and *B. suis* biotype 4 is probably enzoonotic in most Canadian caribou and reindeer herds (Forbes 1991).

For diseases such as tuberculosis and brucellosis, climate change effects are likely to be mediated through changing agricultural activities and changing ranges of wild reservoirs, which will alter contact between wildlife and livestock. Warming in northern tundra regions of Ontario, with changes in caribou movements, will likely change the dynamics of the disease and potential for human exposure. However, southward movement of infected animals is unlikely to reach agricultural areas in Ontario for the foreseeable future.

2.1.5. Chronic Wasting Disease

Chronic wasting disease (CWD), one of the transmissible spongiform encephalopathies, is a prion-related disease. It was first diagnosed in 1978 in captive mule deer (*Odocoileus hemionus*) in Colorado and was later recognized in free-ranging elk, mule deer, and white-tailed deer in Colorado and Wyoming and in farmed elk in Saskatchewan, South Dakota, Nebraska, Montana, Colorado, and Oklahoma (reviewed in Williams et al. 2001). While the mode of transmission is unknown, there is strong evidence for lateral and possibly maternal transmission. Concentration of deer and elk in captivity, or as the result of supplemental feeding, may increase the likelihood of transmission between individuals. Most recently, CWD has been reported in free-ranging deer harvested in South Dakota in 2001 (Holland 2002) and in Wisconsin (Wisconsin DNR 2002, electronic access), the first time the disease has been identified east of the Mississippi River. Its arrival in Wisconsin places 20 million white-tailed deer in the eastern United States and Canada at risk.

Climate change, interacting with habitat changes, will affect the distribution of the disease through impacts on the distribution and abundance of white-tailed deer.

2.2. Environmental Contamination

2.2.1. Environmental Fungal Contaminants

Fungi can affect animals in two ways: by causing disease directly or by producing toxins (mycotoxins) in feed that the animals ingest. Both of these will likely be affected by climate changes.

a) Mycotoxins

The worldwide contamination of foods and feeds with mycotoxins is a significant problem. These secondary metabolites of moulds have adverse effects on humans, animals, and crops and result in illnesses and economic losses. Some moulds are capable of producing more than one mycotoxin, and some mycotoxins are produced by more than one fungal species (Hussein and Brasel 2001). In animals and humans, exposure to mycotoxins is mostly by ingestion, but it also occurs through skin and inhalation (Peraica et al. 1999). Mycotoxins have various acute and chronic effects on humans and animals (especially monogastrics), ranging from depressed growth to cancer and changes in hormonal activities, depending on the species and individual susceptibility. Ruminants however, have generally been more resistant to the adverse effects of mycotoxins because the rumen microbiota can degrade mycotoxins (Hussein and Brasel 2001).

Mycotoxin contamination occurs when feed and food sources are stored at high humidity, conducive to fungal growth. While production loss due to reduced feed intake or subclinical intoxication is the most likely outcome, sickness and death can occur. According to the Canadian Food Inspection Agency, "The mycotoxins of major concern are the trichothecenes (including deoxynivalenol (DON), nivalenol (NIV), T-2 toxin and HT-2 toxin), zearalenone (ZEN), the fumonisins (FB) (predominantly fumonisin B1 (FB1)), the ochratoxins (predominantly ochratoxin A (OA)), and ergot" (Charmley and Trenholm 2002, electronic access). In the same report, the major health effects in animals were reported as "feed refusal and vomiting (DON); impaired reproductive function and reduced fertility (ZEN, DON, T-2 toxin); nephrotoxicosis (OA, FB); neurotoxicosis (FB); lung disease (FB); hepatotoxicosis (FB); cancer (AF, OA, FB), and death (AF, T-2 toxin, FB, OA). Research has demonstrated subtle effects of mycotoxin contamination, including reduced immune function with compromised resistance, and reduced animal performance (DON, AF, T-2 toxin, OA)."

Aflatoxins, which have been linked to liver cancer, are metabolites produced by various fungi (*Aspergillus* spp.) growing on spoiled feeds, predominantly corn, sorghum and peanuts. Currently these toxins occur most commonly in warm humid regions of the southeastern United States. Warm day and night temperatures (>21°C) with high relative humidity and significant insect damage are major risk factors. During drought years, widespread contamination has occurred throughout the corn and cotton-growing areas of the United States (Osweiler 2000).

Warm, wet seasons favour the growth of *Claviceps purpurea*, a fungus that infests cereal rye, barley, triticale and wheat, as well as many grasses. Ingesting large quantities of seed heads infested with the sclerotia (ergots) causes ergotism in cattle, sheep, pigs, horses (*Equus equus*), dogs and birds. Ergotism is characterized by a wide range of neurological signs and gangrene of the extremities. Cycles of mycotoxin production are closely related to weather conditions that favour ergot infestation, and large regions may be involved during specific weather conditions and years (Osweiler 2000).

Fumonisins are mycotoxins produced by *Fusarium moniliforme* growing on mouldy corn grain that has been affected by rain while on the stalk or stored wet. Horses and pigs are highly susceptible to poisoning. Ingesting fumonisins B-1 and B-2 can cause equine leukoencephalomalacia in horses and donkeys and pulmonary edema and hepatopathy in swine. Fumonisins have been reported as common contaminants of corn-based feeds world-wide, including in the United States.

The non-macrocyclic trichothecene mycotoxins (T2 toxin, deoxynivalenol, diacetoxyscirpenol), produced by various fungi including *Fusarium* spp., cause various symptoms including vomiting, feed refusal and anorexia in pigs. Reports of field contamination are principally from the northern United States and Canada, in cool, temperate regions. A recent report from the Canadian Food Inspection Agency found that 84% of barley and 47% of oat samples collected in Eastern Canada were contaminated with this toxin (CFIA 2001b, electronic access).

Zearalenone is an estrogenic mycotoxin produced by *Fusarium graminearum* and *F. roseum*, growing on mouldy corn or barley. Clinical signs of poisoning include vulvovaginitis and infertility in pigs and reduced conception rates in cattle. Regions of concern include the corn- and wheat-producing regions of Canada and the northern and central United States. Moderate temperatures and humid weather conditions favour production of zearalenone. In 1999, in some Ontario counties, up to 20% of cobs were infested (OCPA 1999, electronic access).

Fungal contamination of stored hay can also predispose cattle to mycotic rumenitis and mycotic abortions.

In general, environmental conditions, including temperature and humidity, influence the presence of fungi and the production of toxins. In particular, drought that results in crop damage, followed by warmth and high humidity, will result in increases in fungal growth and mycotoxin production. Since these are conditions that are likely to become more common under climate change, animal diseases related to mycotoxins can be expected to become more common.

b) Mycotic Infections

Conditions favouring dermatophilosis (mycotic dermatitis, *Dermatophilus congolensis*), a fungal dermatitis of sheep, cattle and horses, include heavy rain, followed by warm, humid weather, which reduces the barrier function of skin to infection (Radostits et al. 2000). Predicted climate changes of increased rainfall and higher temperatures in parts of Ontario could result in an increased incidence of dermatophilosis.

Ringworm is the common name for superficial skin infections caused by fungi. These are associated with encrustations, hair loss and irritation. Transmitted by direct contact among animals, including people, these skin infections are more common when temperatures are warm and the air is damp.

Blastomycosis, a systemic fungal infection caused by *Blastomyces dermatitidis,* affects primarily dogs and people, although several species of wildlife are also susceptible (Legendre 1998). At least one case affecting a cat has been reported in Ontario (McEwen and Hulland 1984). There is a well-defined endemic range in the eastern United States and Canada, usually in proximity to bodies of water such as rivers, creeks or lakes, although the epidemiology is not fully understood. The Kenora catchment area in northwestern Ontario has the highest reported incident case rate in North America (Dwight et al. 2000). Soil is believed to be the reservoir for the fungus, and a specific set of environmental conditions is required for proliferation. Rain or heavy dew appears to facilitate the release of infectious spores from mycelial growth in the environment. Most sufferers acquired the disease by inhaling spores.

Histoplasma capsulatum, a soil-borne dimorphic fungus that causes systemic disease in cats (*Felis domesticus*) and dogs, has a similar area of endemicity in eastern North America. The organism can survive wide fluctuations in environmental temperature and prefers moist, humid conditions and soil containing nitrogen-rich organic matter such as bird and bat excrement (Wolf 1998).

Cryptococcus neoformans has a worldwide distribution and infects cats, dogs, humans and a variety of wild mammals. The mode of infection is likely airborne (Jacobs and Medleau 1998). Since 1999, Vancouver Island has been experiencing an epidemic of cryptococcal disease, including pneumonia and systemic disease, related to a variant (*c. neoformans var gattii*) which previously had been reported only in Australia, (Drebot et al. 2000, HC-PPHB, 2002, electronic access). Dogs, cats, llamas (*Lama glama*), wild porpoises, a ferret (*Mustela nigripes*) and more than 50 people have been infected since 1999. More than 40 animals have died, including porpoises, as well as two people. Importation of tropical plants and climate warming are thought to be contributing to this increase. The mean annual temperature at the weather station in Nanaimo, in the affected area, has increased 2°C between 1948-2000. How much of this is due to the "heat island effect" associated with urbanization and how much is due to general warming is not clear. This disease has not been reported in Ontario, but similar conditions under some climate change scenarios could result in a similar epidemic in this province.

Clearly, fungal infections are more common under wet, warm conditions. If winters become warmer and more damp, then these infections could become more common.

2.2.2. Environmental Cyanobacteria

Toxic cyanophytes (cyanobacteria or blue-green algae species) can form dense blooms in shallow, warm, slow-moving or still fresh water bodies. They liberate toxins that can cause massive hepatic necrosis and severe gastroenteritis in many species, particularly cattle and dogs (Radostits et al. 2000). Animals are not more sensitive than people to the effects of the toxins; they are simply not as concerned with the way water looks or smells before they drink it. Although we don't know the extent to which cyanobacterial blooms occur across Canada, we do know they are quite prevalent in the Prairie provinces (HC-HECSB 2001, electronic access). These blooms typically occur in late summer to autumn and are associated with high water temperatures, shallow water, sunny weather, and high nutrient concentrations caused by fertilizer runoff or manure or human sewage contamination (Radostits et al. 2000). Changes in rainfall patterns (for instance, longer and more frequent drought periods, followed by brief but very heavy rains), increased temperature, and greater storm frequency causing agricultural runoff, could lengthen the time that conditions are best for cyanophyte blooms, thus increasing exposure opportunities for susceptible animals.

2.2.3. Environmental Bacterial Contaminants

a) Listeria and Clostridium

Mammals, birds, fish, crustaceans and insects have all been shown to carry *Listeria monocytogenes*; these intracellular, aerobic bacilli, are the causative agents of listeriosis in both people and animals. In animals, the disease occurs sporadically and is associated with reproductive (abortions, premature births) or neurological signs ("circling disease"). Reservoirs include domestic and wild mammals, fowl and humans; there is asymptomatic fecal carriage in humans (5%) and animals. Infection of foxes produces an encephalitis simulating rabies. The pathogen survives well in soil, water, food, and feces. Seasonal use of silage as fodder is often followed by an increased incidence of listeriosis in animals (HC-PPHB 2001b, electronic access).

Clostridium botulinum are spore forming, anaerobic bacteria that produce a neurotoxin under anaerobic conditions. Although botulism toxin can affect a wide variety of animals, including people, the most dramatic botulism outbreaks in animals in Canada have been in birds. Dieoffs of hundreds of thousands of birds have been reported over the years, associated with Type C botulism in prairie wetlands. As well, the Canadian Cooperative Wildlife Health Centre has reports of at least two outbreaks in Ontario (Leighton 2000, electronic access). Type E botulism, which tends to be associated with marine and freshwater environments, has been described for birds and people; recurrent epidemics in piscivorous birds in Lake Erie are described later. Reservoirs for the pathogen include soil, water, and intestinal tracts of animals (HC-PPHB, 2001b, electronic access).

Increased moisture levels during harvesting and storage or forages may be associated with increased proliferation of environmental pathogens such as *Listeria monocytogenes* in wet spoiled hay bales or improperly fermented silage. Forage botulism, associated with *Clostridium botulinum* growth and production of type B toxin in spoiled stored forages, is most likely to occur if the forage is very succulent or is wet when it is made.

b) Leptospirosis

Leptospirosis is a zoonotic disease of worldwide significance. It affects all mammals and is caused by infection with pathogenic serovars of the genus *Leptospira*. Serovars are maintained in nature by numerous subclinically infected wild and domestic animal reservoirs that serve as a source of infection and illness for humans and other incidental animal hosts. Up to 40% of cattle herds in Ontario have evidence of exposure to leptospira, however, the disease tends to be sporadic. Cattle can experience systemic disease, decreased milk production, and abortion. The disease is also sporadic in horses, which can experience abortion and periodic ophthalmia. Leptospiral infection in dogs can cause severe systemic disease characterized by vascular, liver and kidney damage that may cause death; animals that survive may act as reservoirs, with long-term colonization in the kidney and persistent shedding of leptospires in urine for months to years. Dogs are often vaccinated, which protects against disease but not shedding.

Transmission of leptospirosis among maintenance hosts is often direct. However, infection of incidental hosts is more commonly indirect, by contact with areas contaminated with the urine of maintenance hosts. Environmental conditions are critical in determining the frequency of indirect transmission. Leptospires require adequate moisture, moderately warm temperatures, and pH-neutral or alkaline water to survive; survival is brief in dry soil or at temperatures below 10°C (Bolin 2000). Therefore, leptospirosis occurs most commonly in the spring, autumn, and early winter in temperate climates. In all species, epidemics of leptospirosis are associated with flooding, presumably because of the increased exposure to water contaminated with animal urine and a concentration of rodents and other reservoir hosts.

In 2000, Ontario had a marked increase in diagnosis of clinical leptospirosis in dogs, part of a general increase observed in the last three years (Prescott et al. 2002). Cases occurred in the spring and fall; major serovars identified were *autumnalis, bratislava, grippotyphosa* and *pomona*. This shift away from the more classic serovars infecting dogs (*canicola* and *icterohemorrhagiae*) likely is a result of intensive past vaccination against these serovars and a general increase in leptospirosis in urban wildlife reservoirs (raccoons and skunks). The large numbers of cases in 2000 was also related to the warmest and third wettest fall of the past decade in Ontario.

Reports of increases in rat populations in Canadian cities (Stevenson 2002) are also of concern, as these animals can be chronic shedders of leptospiral organisms. Warmer, wetter winters could result in an increase in rat populations and thus increase prevalenceof leptospirosis.

c) Enteric Diseases (E. coli, Salmonella, Yersinia, Campylobacter)

Many enteric bacteria are carried by livestock, pet animals such as dogs and cats, and wild populations of animals that are of minor clinical importance for those species but cause serious disease in people. Most often, these zoonotic pathogens are recognized as causing food- and waterborne diseases in people; therefore they are described more fully in Part B of this report. However, climate change may influence the carriage and shedding rates of these organisms in animals and the survival of the pathogens in the environment. Incidence of diseases caused by enteric pathogens transmitted by fecal-oral routes, such as *E. coli, Salmonella, Yersinia* and *Cryptosporidium,* may be affected by environmental conditions, particularly weather changes (temperature, precipitation). Enterohemorrhagic *E. coli* that produce shiga-like toxins (also referred to as *vero-toxins*) are present in the gastrointestinal tract of ruminants and may cause sporadic disease in young calves. However, they are most important as human pathogens (Harmon et al. 2000). The prevalence of *E. coli* O157:H7 in cattle feces increases in warmer months, and contaminated drinking water likely serves as a common source for spread among livestock.

Salmonellosis is an important enteric and systemic disease in domestic livestock and is also a potential zoonosis and foodborne disease worldwide. Spread is through fecal contamination of the environment, primarily feed and water supplies (including streams, rivers and pastures) (Radostits et al. 2000). Environmental temperature and wetness are important as *Salmonella* are susceptible to drying and sunlight.

An exception to the above enteric infections is colibacillosis, a diarrheal disease caused by *Escherichia coli,* which occurs in all species of newborn farm animals and is a major cause of economic loss in this age group. Among the identified risk factors are changes in weather; wet, windy conditions and cold temperatures commonly precede outbreaks of diarrhea in beef calves. Higher mortality occurs in dairy calves exposed to hot environmental temperatures, and high temperatures may precipitate outbreaks (Radostits et al. 2000). Close confinement during storms or inclement weather may result in stress and disease outbreaks.

d) Q Fever

Exposure to *Coxiella burnetii,* the intracellular rickettsial-like organism that causes Q fever, is widespread in domestic livestock in Ontario (Lang 1989, Lang et al. 1991). Although the disease is seldom reported in cattle (despite a high serological reactor rate to *Coxiella*), abortion outbreaks have occurred in sheep and goats in Ontario in the past few years (P. Menzies, pers. comm). Q fever is reported sporadically in people, in which it may have many clinical manifestations, usually appearing as a flu-like illness; more serious cases have signs relating to endocarditis, hepatitis, and/or pneumonia (Le Ber 1992). In infected animals, which are not always sick, the organism may be shed in the fluids and tissues expelled at birth and in milk. Transmission is then through inhaling aerosolized particles and by contact with contaminated straw and dust. In some areas, horses, dogs, cats, camels, bison, pigeons, geese and rabbits have been infected. Many species of ticks and flies appear to transmit the organism in the wild (Marrie 1990).

Several characteristics suggest that climate change could affect the epidemiology of this disease in Ontario. In Europe and elsewhere, large outbreaks have occurred when infected materials have been transmitted through windborn dust (Salmon and Howells 1982, Dupuis et al. 1987, Tissot-Dupont et al. 1999). Antibody prevalence in deer has been shown to follow a seasonal variation, peaking in mid-winter. Marrie et al. (1985) demonstrated that *C. burnetii* was an important cause of community-acquired pneumonia in the Maritime provinces of Canada. Combined with our understanding of the general biology of Q fever, these reports suggest that the disease is vulnerable to climatic variation, although it is not clear in which direction the effects might be. Increased warmth and humidity could increase transmission through ticks; on the other hand, droughts and generally lower rainfall predicted under some climate change scenarios, have been associated with large outbreaks of Q fever.

e) Anthrax

Anthrax is a bacterial disease, caused by *B. anthracis,* which occurs worldwide, including certain foci in Canada. Infected animals often die acutely; cattle and sheep may die in 1-2 hours after the disease onset. Horses and pigs may live for a day or two. Outbreaks generally occur during dry summer months following periods of heavy rain or floods. Prolonged rain or flooding preceding outbreaks promotes runoff and eventual pooling of standing water (Gates et al. 2001). Drying weather, which often precedes outbreaks, may concentrate spores in depressions because such storage areas would be the last to experience evaporation. The disease is diagnosed sporadically across Canada. In 2001, for instance, three livestock premises (in Alberta, Saskatchewan and Manitoba) were quarantined for anthrax. As well, more than 90 bison died of anthrax in Wood Buffalo National Park (Alberta) (CFIA 2001a, electronic access). Anthrax could occur in Ontario as well, particularly given changes in rainfall patterns expected under climate change scenarios.

2.2.4. Environmental Parasitic Infestations

Because many parasites spend at least part of their life cycle outside the animals they infect, they are vulnerable to weather and climatic changes (seeTable A.9 in Part A of this report). Many animal parasites that concern us cause few disease problems in their definitive hosts; some examples are *Baylisascaris procyonis*, an intestinal roundworm of raccoons and *Toxocara canis* and *T. felis* which infect dogs and cats, respectively. However, the larvae of these roundworms can cause diseases in people: visceral larva migrans, in which the wayward larvae wander about the viscera of unsuspecting children, and ocular larva migrans, in which the larvae exit the body from the eye. The raccoon roundworm can even migrate into the brain. Waterborne protozoal infectious agents that are common in Ontario include *Giardia* spp. and *Cryptosporidia*. Most infections are acquired through drinking from contaminated fresh water bodies such as streams, lakes, and rivers and thus are vulnerable to climatic influences. Although a wide variety of animals can be infected by and carry these parasites, they are of greater concern as human pathogens than as pathogens in other species.

Protozoal parasites are similarly unremarkable as disease agents in the animals that serve as reservoirs for human infection. Canada has the distinction of having had the largest reported water-associated outbreak of *Toxoplasma gondii* (in Victoria), caused by a combination of watershed contamination and heavy rainfall. The definitive hosts for *Toxoplasma gondii* are cats, where this protozoal parasite causes minimal discomfort as an intestinal parasite. Once the unsporulated oocysts are shed into the environment, they require at least 2-3 days to sporulate and become infective. Although *T. gondii* can infect a wide variety of species, it is generally of greater concern in people than in animals and is discussed in Part B of this report. Its chief economic importance is on sheep farms, where it can cause abortions and neonatal death. Almost 100% of sheep flocks in Ontario have had some exposure to the parasite (Waltner-Toews et al. 1991).

Because of the necessary maturation of *T. gondii* oocysts in the environment, the occurrence of this disease is vulnerable to any changes in climate. Infection appears to be more common in warm, humid areas, rather than in cool, dry areas. High rainfall tends to be associated with infection in sheep at pasture (Radostits et al. 2000, Dubey and Beatty 1988). Milder winters allow for greater survival of feral cats and higher levels of environmental contamination. Extreme rainfall events promote "flushing" of oocysts into water systems.

2.3. Vectorborne Diseases

In general, successful transmission of vectorborne diseases requires: 1) a susceptible host population, 2) a stable reservoir of the disease, 3) a stable population of a vector-competent species, and 4) a climate supportive of the development of the parasite. Climate may affect vectorborne diseases by affecting the vectors, the disease organisms, the host, transmission pathways, or, more likely, some combination of these. Indeed, biodiversity, which is itself impacted by climate changes, serves important functions in modifying the ecology and epidemiology of vectorborne diseases (see Table E.4, Appendix 2) (Ostfeld and Keesing 2000a).

By definition, vectorborne diseases possess a vector stage, usually an insect, acarid, mollusk or crustacean that is *poikilothermic* (cold blooded) and hence is especially sensitive to changes in climatic variables. Temperature and humidity are the components of the macroclimate that are likely to have the most important direct effect on vector biology and ecology. However, many vectors demonstrate behavioural preferences for particular microclimates, and the impact of climate change on microclimate environments has only occasionally been considered. Climate change can have a range of potential direct and indirect effects on vector biology and on interactions between vectors and the arthropod-borne disease viruses (arboviruses). These interactions are highly complex, and the outcomes can be difficult to predict. These interactions are likely to be profoundly influenced by climate change (Mellor et al. 2000).

As arthropod vectors expand their geographic range, various clinically important vectorborne organisms will move with them. This is of particular concern to clinicians, as the diseases caused by these organisms will be unfamiliar to them and may go undiagnosed, due to either the absence of a clinical suspicion or because of their subclinical nature. Some may be misdiagnosed and inappropriately treated, while others are zoonotic, leading to risk of serious disease in their owners or other humans (Irwin 2002). In addition, exotic disease outbreaks may become more common in companion animal practice as pets increasingly travel with their owners.

Arboviral infections in animals that are affected by climate and are of greatest concern are those that cause encephalitis in horses. These include the alphaviruses associated with western (WEE) and eastern equine encephalitis (EEE); the flaviviruses St. Louis encephalitis (SLE), West Nile (WNV), and Powassan encephalitis (POW); and the bunyaviruses classified as California viruses, including LaCrosse virus and snowshoe hare virus (CDC, 2001, electronic access). These diseases are a concern not just because of their effects on horses, but also because people can get them via mosquitoes.

Viruses borne by *Culicoides* spp. (biting midges), including bluetongue virus and epizootic hemorrhagic disease virus, are of lesser concern in Ontario but are included in this report for sake of completeness. Of these diseases, bluetongue, WEE and EEE are on the federal Health of Animals Act reportable disease list. While EEE has been diagnosed sporadically in Ontario in the past, bluetongue has not. Indeed, bluetongue was last diagnosed in Canada in 1998, in southern British Columbia (CFIA 2001a, electronic access).

While many of the vectorborne diseases affecting animals are viral, some of particular importance for Ontario, notably Lyme disease, are bacterial.

2.3.1. Climate Effects on Mosquitoborne Diseases

The ecology, development, behaviour and survival of mosquitoes and the transmission dynamics of the diseases they transmit are strongly influenced by climatic factors.

a) Western and Eastern Equine Encephalitis Virus

The eastern equine encephalitis (EEE) virus and western equine encephalitis (WEE) virus are arthropodborne alphaviruses. This group of viruses can cause severe illness in horses and humans; the case fatality rate for EEE in horses is 75-90%; for WEE it is 10-15% (Keane and Little 1987). EEE virus cycles between wild birds and *Culiseta melanura* mosquitoes. WEE also circulates in wild birds and is transmitted primarily by *Culex tarsalis*, although it has been isolated from at least nine mosquito species (Artsob 1981). Infection with either virus does not generally cause illness in birds. The vectors are associated with irrigated agriculture and stream drainages (CDC-DVID 2001, electronic access).

In 1972, 30 horses died of EEE in the Eastern Townships of Quebec; the outbreak died out in October of that year, after first frost. EEE antibodies and/or virus have been demonstrated in passerine birds in Long Point and Prince Edward Point, Ontario. The last reported case of EEE in a horse in Ontario was in 1992 (Carmen et al. 1995).

From 1935-38, more than 60,000 horse cases of WEE were reported in Saskatchewan and Manitoba. About 15,000 horses died in 1937-38 alone. After vaccination of horses became common, disease rates dropped. Outbreaks in the 1970s and 1980s in Manitoba and possibly in northwestern Ontario generally involved dozens, rather than thousands, of animals (Artsob 1981).

The ornithophilic mosquito, *Culiseta melanura*, is the enzootic vector, transmitting EEE virus between passerine birds and more rarely from birds to equids or humans, which are typically considered "dead-end" hosts, although the level of viremia in some horses exceeds the minimal titre considered necessary to infect mosquitoes subsequently feeding on them (Calisher 1994). Epizootics begin in swampy habitats and move outward when viremic birds move locally or migrate further away. Transfer of virus occurs from the enzootic vector to epizootic vectors (including *Coquillettidia perturbans*, *Aedes vexans*, *A. canadensis*, *A. sollicitans*), which more commonly feed on mammals. More recently, the introduced *A. albopictus* mosquito has been involved in the transmission cycle of EEE in Florida.

The distribution and prevalence of WEE and EEE in Ontario are not well defined, but they are apparently rare. No antibodies to either virus were detected in a serological survey of 725 sera from several wild mammal species in Ontario (Artsob et al. 1986).

The current range of enzootic EEE virus is the American Great Lakes region and Atlantic and Gulf coasts, including upstate New York, southwestern Michigan, and northeastern Indiana (Calisher 1994, Moore et al. 1993, Carmen et al. 1995). WEE virus has been reported in the Lake Superior area of Ontario, but it is more frequent farther west. Disease in humans and horses has historically been centred in Saskatchewan and Manitoba, where one vector, *Culex tarsalis*, is widely distributed in agricultural areas. Other mosquito genera capable of transmitting this virus include *Aedes, Anopheles, Coquillettidia, Culex* and *Culesita.* Because the virus replicates effectively in birds, they become amplifiers of the virus and important sources of infection for mosquitoes. House sparrows (*Passer domesticus*) and house finches (*Carpodacus mexicanus*) are important amplifying hosts in western Canada (Leighton 2001, electronic access). This virus also infects snowshoe hares (*Lepus americanus*), garter snakes (*Thamnophis* spp.), and leopard frogs (*Rana pipiens*). These birds and other animals are widespread in Ontario.

High rainfall in the preceding fall, and in June and July, contributes to increasing swamp sizes and available mosquito habitat. This, combined with unpredictable changes in north-south winds patterns, will have implications under future changing climate.

b) West Nile Virus

West Nile virus (WNV), a flavivirus of the Japanese encephalitis group, was first isolated in the West Nile province of Uganda in 1937 (CDC-DVID 2002, electronic access). Until 1999, the disease was reported only in Europe, the Middle East, western Asia, and Africa. The virus was first found in North America in association with an outbreak of encephalitis in New York City and surrounding area in late August and September 1999 (Lopez 2002). At first it was suspected to be a related virus which causes St. Louis encephalitis (SLE) (St. Louis encephalitis is discussed in more detail in Part B of this report). In 2001, the virus was detected in dead birds in southern Ontario. A cycle of transmission between wild birds and native mosquitoes has evidently become established in North America, with migratory birds likely playing an important role in the natural transmission cycles and spread. Although it is not known when and how West Nile virus was introduced into North America, international travel of infected persons to New York or transport by imported infected birds may have played a role. Like SLE, WNV is transmitted principally by *Culex* mosquitoes but can also be transmitted by *Aedes, Anopheles*, and other species (Ellis 2001, electronic access).

In 2002, clinical disease and deaths were confirmed in nearly 200 species of birds, reptiles and mammals including dogs and cats and many exotic species at zoo collections. The outcome of infection in horses varies from aymptomatic infection to temporary neurologic deficits to fulminating fatal encephalitis (Ostlund et al. 2000). Humans and horses are generally considered to be tangential dead-end hosts for WNV. Both wetland and terrestrial birds may participate in the natural virus cycle. Birds of the Corvidae family, including American crows (*Corvus brachyrhynchos*) and blue jays (*Cyanocitta cristata*), have been seen to die in large numbers and have been used to track the spread of the North American epidemic. Late in the 2002 season, morbidity and mortality were also reported in owls, hawks, and other birds of prey. It is not clear how this mortality is affecting the population dynamics of those birds and the ecosystems in which they occupy important niches.

Climatic conditions, including a mild winter coupled with prolonged drought and heat waves, may have played a role in the initial outbreak (Epstein 2001), favouring breeding of the principal vector, *Culex pipiens*, in urban environments and accelerating virus development in mosquitoes. Recent studies have confirmed that elevated temperatures increase the vector competence of *C. pipiens* for WNV (Dohm et al. 2002), possibly contributing to the explosive transmission of this virus in New York during the summer of 1999, a year of near-record-high temperatures. The persistence of the virus, and its spread to other states and Canada since 1999, confirms that WNV is now endemic in North America and that its geographic range probably will continue to expand. WNV surveillance in Ontario began in 2000, and the virus was confirmed as causing wild bird mortality in southern Ontario in 2001 and 2002. The virus may be able to overwinter in Ontario, as suggested by the detection of a case early in the transmission season (crow found in Peel,

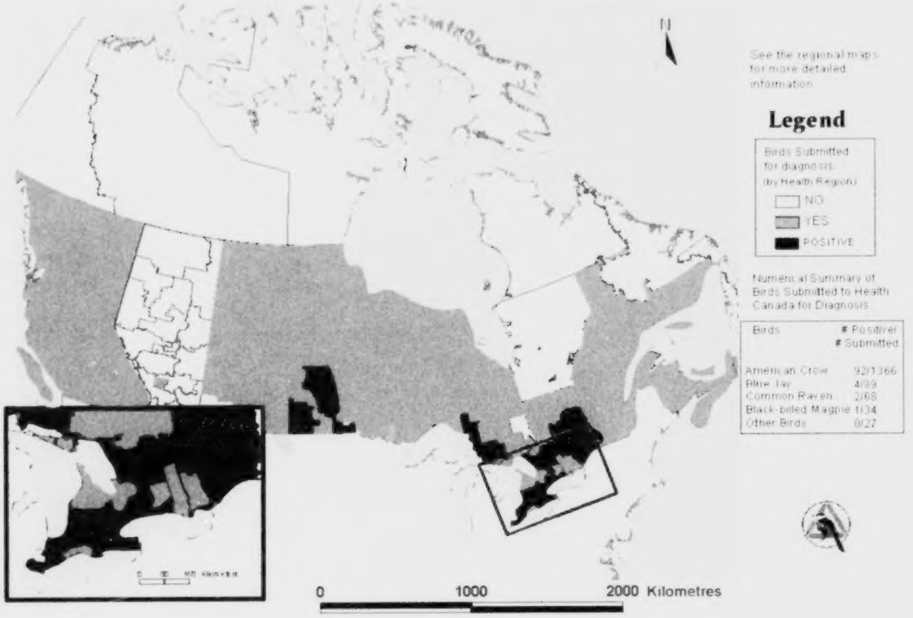
N
See the regional maps for more detailed information
Legend
Birds Submitted for diagnosis (by Health Region)
NO
YES
POSITIVE
Numerical Summary of Birds Submitted to Health Canada for Diagnosis
Birds
# Positive/ # Submitted
American Crow 92/1366
Blue Jay 4/89
Common Raven 2/68
Black-billed Magpie 1/34
Other Birds 0/27
0
1000
2000 Kilometres

Figure C.2. *Dead birds submitted for West Nile virus diagnosis, cumulative 2002 data as of August 2002.*

May 19, 2002) (OMHLTC 2002, electronic access). The spread of the disease in Canada is underway as evidenced by several birds dead of the disease in Winnipeg, MB, in July 2002, and in Quebec and Saskatchewan in August 2002 (MH 2002, electronic access; Figure C.2).

The emergence of WNV is interesting in that its geographic distribution in 2001 in Ontario is similar to that of the SLE virus in 1975. (Artsob 2002, pers. comm.). Both viruses use the widespread *Culex pipiens* as their principal endemic vector; it is suspected that climate factors influence the distribution of the virus over the range of the vector. Since WNV is new to Ontario, little is yet known about its potential spread. The rapid spread of the virus in the United States and Canada and the widespread distribution of the vector in Ontario suggest that the virus could become widely established as well. Climate change projected for Ontario may contribute to the establishment and spread of WNV. As for SLE, local conditions that promote mosquito survival and multiplication include warm but not extremely hot temperatures, adequate humidity, and standing warm water for larval survival. Milder winters and longer summers will extend the transmission season, promote overwintering, and enhance survival of reservoir populations.

c) California Encephalitis Viruses

Snowshoe hare (SSH) and Jamestown Canyon (JC) viruses are mosquito-transmitted Bunyaviridae. SSH, which cycles naturally among mosquitoes (mostly *Aedes* spp.) and small mammals such as snowshoe hares, is widely distributed in Canada; indeed, surveys have suggested that almost 45% of horses in Ontario have been exposed to it. The virus may be associated with mortality in snowshoe hares, but confirmed disease is rare, with only one confirmed case in Saskatchewan, reported in 1983 (Heath et al. 1989). The JC virus, which has been identified in Ontario, Alberta, and Saskatchewan, cycles between mosquitoes and white-tailed deer and possibly moose (*Alces alces*). However, no cases of disease have been reported.

2.3.2. Effects on *Culicoides*-borne Diseases

Climate and weather have dramatic effects on *Culicoides* populations; thus, the epidemiologies of midge-borne viral diseases are similarly affected. Climate affects the activity or biting rates of *Culicoides*, their dispersal, larval development, adult survival, seasonality and abundance.

In the wild, vector *Culicoides* become infected only by imbibing viremic blood from vertebrate hosts. Subsequent transmission of the virus occurs only through biting.

There is no published evidence that any *Culicoides*-transmitted arbovirus can be transmitted vertically (transovarially) or venereally through its vectors, unlike many mosquito, phlebotomid and tick-transmitted arboviruses.

Factors affecting the distribution of vector and non-vector subspecies of *Culicoides* across North America are poorly understood; thus, the effects of climate change on vector distribution are uncertain. However, increasing temperature has been reported to convert non-vector species of *Culicoides* (for example the Palaearctic midge *C. nubeculosus*) into potential vectors for bluetongue virus through a combination of effects (see the "leaky gut" phenomenon in the general discussion on vectorborne disease). A key aspect of the epidemiology of *Culicoides*-borne arboviral diseases is whether adult midges persist all year or disappear for a period when conditions are unsuitable. If freezing temperatures kill adult midges in the absence of reservoir hosts, then the virus must be introduced from elsewhere for disease outbreaks to occur, either by the carriage of infected midges on the wind or by the movement of infected hosts. Thus, both winter warming and changes in wind patterns could interact to change the epidemiology of these diseases.

a) Bluetongue Virus

Bluetongue virus (BTV) is an orbivirus with 24 known serotypes. Infection causes severe systemic disease characterized by stomatitis, rhinitis, enteritis and lameness in certain breeds of sheep and some species of deer. The major vector is a particular subspecies of *C. variipennis*: *C. variipennis sonorensis*. This subspecies is believed to be the primary vector of BTV in the Americas. Populations of another subspecies, *C. v. variipennis*, are the sole representative of the species complex in the northeastern United States, and the low oral susceptibility rates of *C. v. variipennis* to infection with BTV are a major factor in the apparent absence of BTV transmission in this region (Mellor et al. 2000). In the southeastern United States, including Alabama, BTV may be co-vectored by *C. variipennis* and other species of *Culicoides*, including *C. stellifer*. Further south, in Florida, the Caribbean region, and Central and South America, BTV may be vectored by *C. insignis* and *C. pusillus*. BTV has made periodic incursions into the southern Okanagan Valley in British Columbia, the most recent to appear was BTV type 11 in 1998 (Clavijo et al. 2000). A previous outbreak in 1987 and 1988 was suggested to have resulted from windborne infected *Culicoides* from the United States (Sellers and Maarouf 1991).

b) Epizootic Hemorrhagic Disease Virus

Epizootic hemorrhagic disease virus (EHDV) is also an orbivirus, with 10 known serotypes worldwide. In North America, serotypes 1 and 2 are enzootic, but only EHDV-2 has been recognized to occur in Canada. Infection causes severe systemic disease, very similar to bluetongue, in several species of wild ungulates including white-tailed deer, mule deer, and California bighorn sheep (*Ovis canadensis californiana*). Sheep and cattle are usually subclinically infected, although cattle may develop clinical disease as well. The major vector in North America is *C. variipennis*. Sporadic disease incursions have been reported in the southern Okanagan Valley in British Columbia, the most recent in 1999 (Pasick et al. 2001).

2.3.3. Effects on Tickborne Diseases

Illness in humans and companion animals caused by tickborne pathogens are among the most significant emerging diseases worldwide (Irwin 2002). The agents involved encompass a wide range of bacteria, viruses and parasites, many of which are zoonotic. A wide variety of ticks, depending on species and geographic area, are involved (Table C.1).

a) Lyme Disease

The most common tickborne disease in North America, Lyme disease is a multisystemic illness of humans and domestic animals (principally dogs, occasionally cats), caused by the spirochete *Borrelia burgdorferi*. Lyme disease in dogs is characterized by polyarthritis and more rarely, acute progressive renal failure. The organism is carried by ticks of the genus *Ixodes*, which progress through four developmental stages and three blood meals over about two years. The black-legged tick and the western black-legged tick are the principal vectors in Canada. The western black-legged tick is found in British Columbia, while the black-legged tick is endemic in a small area on the north shore of Lake Erie, in Long Point (Barker and Lindsay 2000). Although juvenile ticks have been reported feeding on more than 100 species of mammals, birds and reptiles, the white-footed field mouse (*Peromyscus leucopus*) is considered the primary reservoir in eastern and central Canada and the United States (Ostfeld and Keesing

Table C.1. *Ticks as vectors for diseases important in Ontario under conditions of climate change.*

Many tick species maintained in sylvatic life cycles with wild mammal and/or avian hosts can vector important diseases that affect various species of wildlife, domestic animals and humans (Allan 2001).

- ***Ixodes cookei,*** which occurs east of the Mississippi and in eastern Canada, is a potential vector for Powassan virus, tularemia, and spotted fever group rickettsia. This tick has been reported on at least 25 species, including domestic animals and man. It is relatively common on woodchucks (*Marmota monax*) but also occurs on raccoons (*Procyon lotor*), skunks, and foxes.
- ***Ixodes scapularis,*** the black-legged tick, occurs in the eastern United States and Canada (Ontario) and is a potential vector for Lyme disease, human babesiosis, human granulocytic ehrlichiosis, tularemia, spotted fever group rickettsia, anaplasmosis and Powassan virus. This tick has been reported on 41 species of mammals (including domestic animals and man) and 57 species of birds, although white-footed mice (*Peromyscus leucopus*) and white-tailed deer (*Odocoileus virginianus*) are the most important hosts of immatures and adults respectively (Allan 2001).
- ***Dermacentor albipictus,*** the winter tick, occurs in the northern, eastern and western United States and Canada. It is a potential vector for bovine anaplasmosis, an exotic disease in Canada. Its primary host is moose (*Alces alces*), although it readily feeds on other cervids.
- ***Dermacentor variabilis,*** the American dog tick, is present in the eastern United States and Canada and has a wide host range, with immatures feeding on small mammals such as the white-footed mouse and adults feeding on larger mammals such as white-tailed deer. This tick is a potential vector for rocky Mountain spotted fever, babesiosis, tick paralysis, anaplomosis, Q fever, tularemia and cytauxzoonosis.
- ***Rhipicephalus sanguineus,*** the brown dog tick, is distributed widely throughout North America, primarily on dogs (*Canis domesticus*), particularly in protected habitats such as kennels, and rarely on coyotes (*Canis latrans*), rabbits (*sylvilagus* spp.), and white-tailed deer. It cannot survive temperatures < 5°C. This tick is a potential vector for canine ehrlichiosis, canine babesiosis, Rocky Mountain spotted fever, tularemia, bovine anaplasmosis, Q fever, tick paralysis and haemobartonellosis.

2000b). White-tailed deer are the most important hosts in this area for adult ticks. Dogs, cats and people are incidental hosts. Direct horizontal spread from dogs and cats to people is unlikely, although pets may serve as sentinels for potential human infection.

The range of the black-legged tick in the United States is expanding, and range expansion and a concomitant local increase in the risk of Lyme borreliosis may also be occurring in Ontario, with established tick populations now detected at Point Pelee National Park and Rondeau National Park (Barker and Lindsay 2000). In a survey of ticks collected from southern Ontario dogs in 1997 and 1998, 9 of 139 submitted black-legged ticks were infected by *B. burgdorferi*, as identified by polymerase chain reaction (PCR) (Banerjee et al. 2000). Two infected mated female ticks were collected from dogs from Mississauga and Etobicoke, suggesting that the black-legged tick may be becoming established in these areas as well. Current evidence suggests that the black-legged tick does not have an established population across southern Ontario; however, these ticks may be dropped by birds migrating from tick-endemic areas to the south. If enough of these adventitious immature ticks are introduced to an area, some may survive to mate and establish a local population. However, given that *B. burgdorferi* is not transmitted through the tick egg, subsequent generations of ticks will not pose a threat until an infected host, such as a migratory bird, introduces the spirochete into the host-vector cycle in that vicinity (Barker and Robbins 2000). The black-legged tick can theoretically become established throughout the parts of Ontario that are south of North Bay and in the Kenora-Rainy River region of northwestern Ontario.

Suitable habitat for expanding populations of the black-legged tick depends on the interaction of many biotic and abiotic factors, including habitat type, seasonal variations in microclimates within habitats (temperature and humidity, that are influenced by vegetational composition, density and cover), and habitat use by suitable hosts such as white-tailed deer (Lindsay et al. 1999). Climatic constraints on life-stage development likely will keep ticks from establishing populations in most of northern Ontario (Lindsay et al. 1995). However, the effects of predicted climate change on this potential distribution are complex. Disease transmission is at least partly dependent on species richness (biodiversity) of terrestrial small mammals, as well as, in some areas, masting cycles of oak trees (*Quercus* spp.) and the gypsy moth (*Lymantria dispar*) (Ostfed and Keesing 2000b, Ostfeld 2002, pers. comm.). Ambient temperatures over

27°C for periods of more than two weeks appear to inhibit disease transmission (Shih et al. 1995). Thus, any climate effects on changing vegetation will interact with effects on the vectors, host populations, and the spirochete.

b) Rocky Mountain Spotted Fever

Rocky Mountain spotted fever (RMSF) is a tick-borne rickettsial disease caused by infection with *Rickettsia rickettsii*. The disease affects dogs and people. In dogs, the disease is characterized by systemic vasculitis with fever, edema, and hemorrhage, with cardiovascular, neurologic and renal damage. The disease occurs throughout the contiguous United States, with the exception of Maine, and has been reported in Western Canada but not in Ontario. Deciduous forests, increased humidity and warmer temperatures are factors associated with the high prevalence of this tick-transmitted disease (Greene and Breitschwerdt 1998). The American dog tick *(Dermacentor variabilis)*, found east of central Saskatchewan, and the wood tick (*D. andersoni*), found further to the west can transmit Rocky Mountain spotted fever. These three-host ticks act as natural hosts, reservoirs and vectors for *R. rickettsi*, and can transmit the rickettsia to their progeny. The primary sylvan cycle occurs between small mammals (chipmunks, voles, ground squirrels) and immature tick stages. Temperature increases along with the complex social and ecological changes that are occurring concurrently may be sufficient to allow the disease to move northward into Ontario.

c) Ehrlichiosis

Canine monocytic ehrlichiosis (CME) is a multi-systemic disease caused by infection of mononuclear white blood cells with *Ehrlichia canis*, resulting in fever, anorexia, weight loss, hemorrhage and neurologic signs. Vertebrate reservoir hosts are limited to members of the family Canidae (coyote, fox, dog). The arthropod vector of *E. canis* is the brown dog tick *(Rhipicephalus sanguineus)* with strict transstadial transmission (Neer 1998). This tick cannot establish in Ontario unless the climate warms substantially; however, the expansion of risk areas in the United States would increase the chances for traveling dogs to pick it up and bring it home to Ontario.

Equine granulocytic ehrlichiosis is a disease characterized by fever, anorexia, hemorrhages and dependent edema. The rickettsia *Ehrlichia equi*, an obligate intracellular pathogen infecting white blood cells, is the causative agent of equine granulocytic ehrlichiosis. It is closely related to or even identical to the agent causing human granulocytic ehrlichiosis and is vectored by the black-legged tick, as well as by the western black-legged tick in western North America. The black-legged tick is a known vector of several diseases in animals, including the spirochete *Borrelia burgdorferi* (see earlier section on Lyme Disease), and *Babesia microti* (a human pathogen). Recently, Drebot et al. (2001) reported the first identification and characterization of an ehrlichia of the *E. equi* genogroup from an black-legged tick collected in Long Point, Ontario.

Endemic black-legged tick distribution in Ontario is restricted to the north shore of Lake Erie; however, alterations in climate might favour population expansions (see earlier section on Lyme Disease and Part B for more information).

2.3.4. Effects on Other Insect Vectorborne Diseases

a) Leishmaniasis

Visceral leishmaniasis is a zoonotic protozoal disease that causes chronic debilitating illness in humans and dogs (Slappendel and Ferrer 1998). Sandflies serve as vectors, and wild and domestic dogs are considered to be the reservoir hosts for the organism. Visceral leishmaniasis is considered to be an exotic disease in North America, although the disease is endemic to parts of Africa, southern Europe, central Asia, and Central and South America. However, in 2000 an outbreak occurred among hunting dogs in the United States and Canada, associated with infection by *Leishmania infantum*. Seropositive dogs were identified in 62 foxhound kennels in 21 states and 2 Canadian provinces, including Ontario, suggesting that leishmaniasis is much more widespread in North American canine populations that originally thought (NCSU News Service 2000, electronic access). Some reports suggest that there are endemic foci in dogs in Oklahoma, Texas and Ohio. Frequent movement and exchange of dogs at shows and hunts along with direct dog-to-dog transfer through fighting have been postulated as potential routes of transmission in this outbreak (Irwin 2002). Sandflies indigenous to these regions have not been investigated for their potential as vectors; hence, the effects of climate cannot be predicted.

Nevertheless, a combination of increased animal movements and climate changes would likely increase the risk of this disease spreading.

2.4. Rodentborne Diseases

Although we generally think of rodent-associated diseases such as plague (*Yersinia pestis*) and hantavirus pulmonary syndrome (HPS) as occuring far from Ontario, anecdotal and newspaper reports of increases in rat populations, both worldwide and in Canadian cities (Stevenson 2002), should give us pause. A few cases a year of HPS have been reported from all four Western provinces in Canada (Drebot et al. 2000). Furthermore, infected mice are known to occur in Eastern Canada. Outbreaks of both diseases have been associated with particular weather patterns, primarily through influencing the dynamics of rodent populations and human-rodent interactions (Parmenter et al. 1999, Enscore et al. 2002). Since we are usually less concerned with the health of rodents than of people, these diseases are discussed more fully in Part B of this report. Leptospirosis is also carried by rodents and should increase in areas where there are increases in standing water and rodent populations. This disease is discussed more fully under the section on bacterial diseases spread through environmental contamination.

3. Diseases Important Primarily for Livestock and Horses

Climate change can affect livestock health and production through changes in the quality and availability of pasture, forage and grain, as well as through effects on the dynamics of infectious diseases.

3.1. Direct Effects of Climate On Livestock

Potential direct effects of climate change on domestic animals include reduced performance (e.g., reduced feed efficiency, growth rate, milk and wool production, reproduction), morbidity, mortality and altered disease susceptibility directly associated with thermal stress (linked to air temperature, humidity, wind speed and thermal radiation) (IPCC 2001, electronic access).

Livestock production also is affected by short-term extreme weather events. Power outages due to extreme weather can affect intensively managed livestock (pigs, poultry, etc.). For example, an ice storm in eastern Canada and the northeastern United States in the winter of 1998 had severe effects on livestock in the region (Kerry et al. 1999). Power outages were of particular concern to dairy farmers; dependence on milking machines, for instance, meant farms without power could not regularly milk their cattle, leaving them vulnerable to mastitis. Inadequate ventilation and low temperatures in barns also made cattle susceptible to pneumonia and other infectious diseases. Although an estimated 80% of dairy operations in Quebec had backup generators already in place (due to frequent outages in the past), only about 20% of large Ontario farms had such generators because farmers had not generally experienced frequent power losses. Many large hog and chicken farms had backup power sources for heating, lighting and ventilation, and although an estimated 140,000 chickens and 8,000 pigs died during the storm, this was a small fraction of the region's 22 million chickens and 4 million pigs.

Heat stress increases water requirements and reduces feed intake and digestibility, decreases growth rates, depresses milk production and reproductive efficiency, and increases embryo mortality. Although brief exposures may have little effect, vulnerability to heat is a concern for unacclimated animals, feedlot cattle near market weight, and high-producing dairy cows. Heat stress is most common during sustained hot weather or acute heat loads imposed by heat waves, particularly if there is little or no opportunity for nighttime recovery (Hahn 1999, Nienaber et al. 1999). When the magnitudes (intensity and duration) of adverse environmental conditions exceed threshold limits with little or no opportunity for relief (recovery), animal functions can become impaired by the resulting stress, at least in the short term. A 1995 heat wave in the midwestern United States resulted in severe livestock performance and death losses, with an estimated $28 million economic loss to farmers. In Rockport, Missouri, more than 4,000 feedlot cattle died (Hahn 1999). Genetic variation, life stage, and nutritional status also influence livestock vulnerability to potential environmental stresses.

Klinedinst et al. (1993) used biological response functions of dairy cattle and temperature increases predicted by three global circulation models (GISS, GFDL, UKMO) to model dairy cow performance in the United

States. The results predicted substantial reductions in dairy cow performance, with a general trend of increasing effect from the northwest to the southeast. For example, milk production of moderate- to high-producing shaded dairy cows in hot/hot-humid southern regions of the United States might decline an additional 5-14% beyond expected summer reductions. However, regions that currently experience little or no mild production decline due to summer heat stress, such as the northeastern United States and Ontario, were also projected to have notable milk production declines. The economic impact may be more significant in these areas as farmers are not accustomed to dealing with heat stress, and unlike in the south, have not adopted environmental modifications such as evaporative cooling or air conditioning. Substantial declines in conception rates for dairy cows were predicted for the eastern and southern United States, with reductions of as much as 36% during the summer season in the southeast.

A higher susceptibility to infections has been observed in cows suffering from heat stress (Webster 1983). The effect on disease resistance or immunoresponsiveness appears to vary with species and breed, duration of exposure, severity of stress and the type of immune response considered. The mechanism of this increased susceptibility is unknown. Limited studies have shown a reduction in white blood cell numbers and alterations in circulating T-lymphocytes in crossbred cattle exposed to heat stress, suggesting the possibility of altered resistance to environmental pathogens following heat stress (Morrow-Tesch et. al. 1996). Other studies, however, have reported no effect of moderate heat stress on cell-mediated immunity, protective value of colostrum or transfer of passive immunity to calves (Lacetera et al. 2002).

Not all effects on livestock are likely to be negative. For fall and winter months, climate change is generally predicted to benefit livestock production in all regions due to reduced feed requirements, increased survival of young, and lower energy costs. These benefits may balance out some of the negative heat stress effects in summer months.

In general, intensively managed livestock such as pigs and poultry are kept in controlled-environment buildings year-round, with microclimate closely regulated for temperature, light and ventilation. Thermal stress can result if building climate control cannot compensate for external conditions, resulting in elevated temperatures and/or humidity that surpass an upper critical limit.

Simulation models have been developed in Britain to assess the potential impact of climate change on intensive livestock systems (Turnpenny et al. 2001). These models integrate data on livestock feeding, livestock thermal balance, thermal balance of controlled environment buildings, and a stochastic weather generator to predict effects on growing pigs and broiler chickens in southeastern England (annual mean temperature 9.6°C, increasing to 11.0°C in the climate change scenario). The model predicted a substantial increase in the frequency of severe heat stress, with a consequent risk of mortality. To offset this increase, it would be necessary to reduce stocking densities considerably or to invest in improved ventilation or cooling equipment. Other effects on production (growth rate, feed intake) were negligible.

A companion model was developed for grazing livestock (dairy cows, beef calves and sheep) at three locations in Britain (Parsons et al. 2001), integrating grass production, livestock feeding, livestock thermal balance, the thermal balance of naturally ventilated buildings, and a stochastic weather generator (annual mean temperatures ranged from 8.1-9.6°C, and increased to 9.4-11.0°C; annual mean rainfall ranged 564 mm-1777 mm and increased to 616 mm-1959 mm). Results predicted that grazing livestock should be able to adapt to the expected climate changes, with a small increase in grass production possibly allowing an increase in total productivity in some cases. Developing similar models may be useful in predicting effects on Ontario livestock.

Estimates of livestock production efficiency suggest that the negative effects of hotter weather in summer outweigh the positive effects of warmer winters (Adams et al. 1998). Under a 5°C increase in temperature, livestock yields fell by 10% in cow-calf and dairy operations in the Appalachia, the Southeast, the Delta states, Texas and the southern plains regions of the United States. For a 1.5°C warming in the same regions, the yield loss was estimated at 1%.

3.2. Indirect Effects of Climate on Livestock

For animals reared extensively (at pasture), including llamas, bison, elk, emus (*Dromaius novaehollandiae*), ostriches (*Struthio camelus*), boars, peafowl (*Pavo cristatus*), backyard or free range swine and poultry, beef, and, seasonally, dairy cows, there may be more direct climate effects. Altered climate may contribute to range shifts in poisonous pasture plant species. Reports from drought-affected parts of the Canadian prairies during summer 2002 suggested a wide range of unexpected outcomes. Some pasture grasses, for instance, appear to take up nutrients differentially, resulting in outbreaks of disease that look like calcium imbalances ("milk fever") in beef cattle, a syndrome normally only seen in high-producing dairy cows. Dust storms moving across the alkaline dirt of dried-up prairie sloughs have been associated with outbreaks of pneumonia in cattle. In extreme conditions of drought, starvation of cattle at pasture can become a serious problem. (C. Waldner 2002, pers. comm.).

Even for more intensively managed animals, many diseases of livestock in Ontario (and indeed, globally) occur seasonally, associated with seasonal weather variability, even if they are not associated with vectors or life cycles outside the species of interest. The relationship may be indirect, resulting from concurrent seasonal changes in feeding, housing, degree of crowding, subsequent stress and opportunities for disease transmission, rather than temperature dependence of the pathogen itself. In some instances, expected climate effects may be positive. For example, an increase in mean winter temperatures may reduce the severity of outbreaks of bovine respiratory disease seen mainly in the late fall and winter. Similarly, risk of hypothermia and subsequent hypoglycemia in newborn animals, particularly lambs, may be reduced.

To the degree that climate change in Ontario affects domestic animal husbandry, alterations in incidence and severity of diseases with an apparent seasonal pattern may occur.

3.2.1. Diseases of Cattle and Horses

a) Mastitis

Climate, housing system, type of bedding and rainfall interact to influence the degree of exposure of dairy cattle to mastitis pathogens (Radostits et al. 2000). High The humidity and high ambient temperatures favour growth of pathogens. Cows in confinement housing with organic bedding materials have the highest incidence of environmental mastitis (including *Streptococcus* spp., coliforms) in the warm, humid months of the year. In drylot systems, the incidence of coliform mastitis may be associated with periods of high rainfall. In general, the quality and management of housing for dairy cattle has a major influence on the types of pathogens and degree of infection pressure. Overcrowding, poor ventilation, and access to dirty ponds of water and muddy areas where cows congregate are major risk factors. Outbreaks of summer mastitis (*Arcanobacter pyogenes*) in pastured cattle in Europe, the United Kingdom, Florida, and elsewhere appear to be spread by biting flies. The incidence is much higher in wet summers and on heavily wooded and low-lying farms where the fly population is high.

b) Enteric Parasites

Strongylid nematodes, including *Ostertagia, Trichostrongylus, Haemonchus, Nematodirus* and *Cooperia* are common pathogenic intestinal parasites of ruminants worldwide (Radostits et al. 2000). However, their importance in Ontario is sporadic, probably limited by climate-related restrictions to survival of overwintering larvae on pasture and the use of anthelmintics. In other, more temperate climatic regions, these parasites can cause significant disease and production loss, and control is complicated by the development of resistance to commonly used anthelmintics.

Life cycle of these nematodes is direct; transmission is by ingestion of infective larvae. Disease risk is determined by factors influencing the susceptibility of the host, the numbers of infective larvae accumulating on pasture, and the numbers of larvae undergoing hypobiosis. Hypobiosis is a form of seasonal arrested development, occuring when the prevailing seasonal temperature and humidity fall to levels that threaten larval survival. This mechanism ensures survival of nematodes when prevailing environmental conditions are unfavourable. Increased winter temperatures may improve egg and larval survival. Rate of larval development is temperature dependent, occurring faster in warmer weather. However, infective larvae have shorter lifespans at higher temperatures, and the number of larvae accumulating on pasture is a balance between these two opposing factors. This pattern has been modelled to investigate trends in different climatic zones and under different husbandry systems.

The barber pole worm (*Haemonchus contortus*) is an important abomasal parasite in sheep and goats, capable of causing heavy losses. Infective larvae develop rapidly in warm, wet conditions, leading rapidly to dangerous levels of pasture contamination. Opportunities for transmission are restricted by larval susceptibility to desiccation and cold.

Nematodes in two subfamilies, Strongylinae (large strongyles) and Cyathostominae (small strongyles or cyathostomes) are important enteric parasites of horses. The life cycle is direct, and survival of eggs and larvae is favoured by shady, moist conditions and moderate temperatures. The extensive use of high-efficacy anthelmintics has greatly reduced the prevalence of large strongyles in many regions; however, the cyathostomes are becoming increasingly important, in part due to relative insusceptibility of mucosal cyathostomes to many drugs and to selection for drug resistance.

c) Equine Protozoal Myeloencephalitis

Equine protozoal myeloencephalitis (EPM) is a neurological disease of horses characterized by incoordination (ataxia), weakness, spasticity and muscle wasting. It was first diagnosed in Kentucky in 1964 and is caused by infection with *Sarcocystis neurona* (rarely *Neospora* spp.) (MacKay et al. 2000). All *Sarcocystis* organisms alternate serially between definitive and intermediate hosts. The definitive host for *S. neurona* in North America is the opossum (*Didelphis virginiana*). The intermediate hosts appear to be raccoons and skunks. Horses probably acquire the infection from opossums. Distribution of opossums closely correlates with the distribution of EPM cases in North America. The opossum has recently experienced a population expansion in southern Ontario, possibly due to climatic factors and habitat availability. Thus, even without possible changes to raccoon and skunk populations already alluded to, infection rates in horses are likely to increase as winters get milder.

d) Equine Monocytic Ehrlichiosis

Equine monocytic ehrlichiosis, also known as Potomac horse fever, is a disease caused by infection with *Ehrlichia risticii* and is characterized by fever, colic, diarrhea, laminitis and abortion. The disease has a seasonal occurrence, associated with pastures bordering creeks and rivers. Recent evidence suggests that trematodes, which use operculate freshwater snails as intermediate hosts, are involved in the life cycle of *E. risticii* (Madigan and Pusterla 2000). Two potential trematode vectors, *Acanthatrium* spp. and *Lecithodendrium* spp., both infected with *E. risticii*, have been found in the intestines of bats and birds. These trematodes are common parasites of bats, birds and amphibians in North America, which use pleurocerid freshwater snails as first intermediate hosts and aquatic insects as second intermediate hosts. The mode of transmission to horses is currently being investigated and may involve consuming infected cercariae in water or metacercariae in second intermediate hosts.

e) Verminous Pneumonia (Lungworm)

Verminous pneumonia in cattle is caused by infection with the bovine lungworm, *Dictyocaulus viviparus* (Radostits et al. 2000). The disease is widespread in temperate areas but is most important in mild, damp regions of the British Isles and parts of western Europe. Immunity develops relatively quickly, but cattle will succumb if exposed to overwhelming numbers of infective larvae while grazing. Larvae are passed in feces; moisture and a moderate temperature are essential for development to the infective stage. Recently, the numbers of outbreaks in cattle in the United Kingdom, Denmark and some other countries have been rising. Reasons for the rising numbers are speculative but include declining use of vaccines; use of highly effective anthelmintics in the first grazing season, which may prevent adequate antigenic exposure; and possible changes in weather patterns. Warm, wet summers give rise to heavier burdens of *D. filaria* lungworms in sheep in the following autumn and winter.

f) Hepatic Fascioliasis

Fasciola hepatica is the most common and important liver fluke of sheep and cattle and has a cosmopolitan distribution, including North America (Radostits et al. 2000). Lymnaeid snails are intermediate hosts, and release the infective metacercaria into herbage. The risk of disease is determined by the number of infected snails in the grazing area which depends on the amount of suitable snail habitat. Snail habitat varies by species from aquatic to damp or wet environments, swampy areas, streams, and springs. The main factors determining the timing and severity of hepatic fascioliasis are those that influence the number of metacercariae accumulating on herbage. In particular, temperature and rainfall affect both the spatial and temporal abundance of snail hosts and the rate of development of fluke eggs and larvae.

g) Foot Rot

The incidence of bovine interdigital necrobacillosis or foot rot (due to *Fusobacterium necrophorum*) is typically higher during wet, humid weather, or when conditions are wet underfoot (Radostits et al. 2000). It would likely increase if those conditions become more common in Ontario.

3.2.2. Diseases of Poultry and Swine

In Ontario, poultry, and to a large extent swine, are raised indoors under intensive conditions. The effects of climate change on these species is thus more likely to be via more direct mechanisms such as heat stress and power-failures after ice storms, than via changed exposure to infectious diseases. Free-range pigs and chickens are more likely to be exposed to changes in disease patterns. However, as recent epidemics of livestock diseases in Europe and the Far East have demonstrated, the systemic nature of intensive rearing makes these animals considerably more vulnerable to large-scale epidemics when the agents do enter the system.

a) Newcastle Disease

Newcastle disease (ND) is one of the most important viral diseases of domestic poultry and occurs in a spectrum from undetected subclinical infections (associated with lentigenic strains) to highly fatal epizootics (caused by viscerotropic, neutrotropic or pneumotropic velogenic strains). Velogenic Newcastle disease (NDV) is a reportable disease in Canada, with significant implications for the poultry industry. Wild waterfowl act as a reservoir for NDV, with clinical disease occurring only rarely (Wobeser 1997). However, in summer 1992, NDV caused epizootic mortality in double-crested cormorants (*Phalacrocorax auritus*) in the Great Lakes region, western Canada, and seven northern American states (Banerjee et al. 1994, Glaser et al. 1999). Based on repeated epizootics in cormorants since 1990, NDV appears to be established in double-crested cormorants (Glaser et al. 1999). The possible transmission of this virus from free-ranging wild birds to domestic poultry poses a significant concern.

3.2.3. Diseases of Farmed Fish

Fish farming, heralded as a new source of income for Prairie farmers who wished to diversify in the 1970s, has largely disappeared with the long-term drought in that area. However, even without such dramatic water loss, aquaculture in Ontario is likely to be affected by climate changes.

The dominant aquaculture species in Ontario is the rainbow trout (*Onchorhynchus mykiss*), a Pacific salmonid that is best adapted for relatively cold water. Increases in temperature would probably result in higher, but not catastropic, losses of this species, due to thermal stress and higher susceptibility to bacterial diseases. Farmed fish appear to become very susceptible to stress and disease if the water temperature rises rapidly; fish can adapt to higher temperatures (to an extent) if they rise gradually.

Even a small change in temperature (as low as 5°C) can promote overt furunculosis in Atlantic salmon fry (*Salmo salar*), possibly due to an enhanced rate of replication or enzymatic production by the pathogen that is not matched by that of the fish's immune mechanisms. Warmer water species, such as tilapia (*Oreochromis niloticus*), are gaining a strong foothold in provincial aquaculture and might be less susceptible to global warming-related effects.

Heavy rainfalls appear to be associated with outbreaks of bacterial gill disease in farmed fish. It is not clear whether this is due to higher amounts of particulate matter washing into the water and irritating the gills (allowing for entry of this ubiquitous pathogen) or to a higher pathogen load in the water.

Interactions among changes in average, maximum, and minimum temperature; changes in the quality and quantity of water available; excessive rainfall runoff; and increased ultraviolet exposure make predicting effects in farmed fish a major challenge. This challenge exists for aquatic systems in general (see below). Some of the diseases that should be monitored include bacterial gill disease (flavobacteria), furunculosis (*Aeromonas salmonicida*), bacterial kidney disease (*Renibacterium salmoninarum*), skin necrosis (*Flexibacter columnaris*), and whirling disease (*Myxosoma cerebralis*).

4. Diseases Important Primarily for Dogs and Cats

Since many of the organisms that affect pet dogs and cats are zoonotic, they have been discussed earlier in this report. A few diseases are of concern primarily for the dogs and cats themselves, and these are mentioned here.

The major mosquito-borne disease to affect dogs and cats in North America is heartworm. Infection of the blood, right ventricle of the heart, pulmonary artery and vena cava with the filariid worm *Dirofilaria immitis* can lead to pulmonary hypertension and right-sided heart failure. Infection is widespread across the United States. Accumulated evidence from veterinary clinic reporting since the 1970s suggests that the parasite moved into southern Ontario and has become endemic (Slocombe 1990, Slocombe and Villeneuve 1993). However, the prevalence of infection is low and is clustered in four regional foci: southern Ontario (Windsor, Sarnia, Lake Erie area), southern Manitoba, the Okanagan Valley of British Columbia, and southern Quebec (around Montreal). Heartworm is vectored by several mosquito genera, including *Aedes* spp. and *Culex* spp. Development of *D. immitis* larvae requires approximately two weeks of temperature at or above 27°C; no development will occur if the temperature goes beneath 14°C. As a result, heartworm disease is seasonally, as well as geographically, limited.

The tick *Rhipicephalus sanguineus* is a vector for *Babesia canis* and *Babesia gibsoni*, haemoparasites causing hemolytic disease of varying severity in infected dogs. Canine babesiosis occurs in the United States, most commonly along the Gulf Coast, and in the southern, central and southwestern states, particularly Arkansas, Arizona, Florida and Oklahoma (Taboada 1998). Outbreaks may occur in localized populations, such as kennels; there is a high incidence of infection in racing greyhounds in the United States. *Rhipicephalus* can also transmit *Hepatozoon canis*, a blood parasite that is associated with gait dysfunction in dogs and can infect coyotes, raccoons, foxes, bobcats (*Lynx rufus*) and ocelots (*Leopardus pardalis*).

Since feral cats serve as reservoirs for many infectious diseases of domestic cats (as well as zoonoses such as toxoplasmosis, discussed earlier), to the extent that warmer winters increase survival rates of feral cats, risk to domestic cats will be affected.

5. Diseases Important Primarily for Wildlife

With few exceptions, most wildlife diseases that concern us and that are vulnerable to climatic change also affect people and/or livestock. This priority is probably more a function of the realities of research funding allocations rather than of the disease realities. In almost all cases, the effects are mediated through the complex dynamics of ecosystems.

Human-caused environmental changes affect wildlife directly through habitat loss, human encroachment into wildlife territory, significant habitat fragmentation, and loss of biodiversity. These alterations in the physical structure of landscapes can interact with climate changes to change ecosystem and disease dynamics significantly.

For instance, Johnston and Schmitz (1997) attempted to model the effects of doubling of atmospheric CO_2 on four mammalian species (elk, white-tailed deer, Columbian ground squirrel [*Spermophilus columbianus*] and chipmunks [*Eutamias* spp.]). Altered thermal conditions alone had little or no effect on the species' distributions, as temperatures were well within physiological tolerances. However, under climate change, species distributions were predicted to shift in response to vegetation shifts, even for widely distributed 'generalist' species such as white-tailed deer.

Habitat changes that increase suitable habitat for adaptable or generalist wildlife species may also affect the level of infection and potential for transmission of diseases associated with these species. Physiological stresses induced by high temperatures, extreme weather events, and ultraviolet radiation can compromise host resistance and increase the frequency of opportunistic diseases. How these factors influence wildlife disease ecology is poorly understood (Daszak et al. 2001).

Examples of animals affected by such habitat changes include raccoons, which are potential reservoirs for rabies, canine distemper virus, leptospirosis, toxoplasma, *Baylisascaris procyonis* and pseudorabies virus (Mitchell et al. 1999), and white-tailed deer, important in the transmission cycles of Lyme disease, meningeal worm (*Parelaphostrongylus tenuis*), and in certain areas of North America, chronic wasting disease and bovine tuberculosis.

Wetlands are particularly sensitive to the indirect changes in regional hydrology that climate change may influence, through changes in air temperature, regional precipitation, surface runoff, snow cover, length of the freezing season, ground water storage, and evapotranspiration (Mortsch 1998). The Great Lakes shoreline wetlands provide important spring and fall migration and staging habitat for waterfowl from the Atlantic and Mississippi flyways, as well as nesting habitat for various species. Some mammal species such as muskrats (*Ondatra zibethicus*) are also dependent on wetland habitat, and the littoral zone of wetlands provides food, shelter, spawning and nursery sites for a wide variety of fish. Under climate change, the two most likely water level scenarios for Great Lakes shoreline wetlands are increased frequency and duration of low water levels or a changed temporal distribution and an increase in seasonal water levels. Either of these scenarios could affect primary productivity; use by wildlife, waterfowl and fish; water quality; areal extent; and diversity (Mortsch 1998).

Because of the complexity of the effects mediated through ecosystem changes and the paucity of information on infections affecting wildlife, only a few diseases are singled out in this report.

5.1. Wild Ruminants

Alterations or loss of habitat as a result of climate change may prove a significant stressor on some populations of wild animals, particularly those with restricted suitable habitat. *Dermacentor albipictus* (also called moose tick, winter tick or elk tick), is known to feed on a variety of ungulates in Canada and to cause mild to severe disease in moose, elk, and caribou. In more severe cases, associated with substantial blood and hair loss and distraction from eating, especially during the winter months, the animals starve to death. Loss of wetland habitat and feed sources will exacerbate these cases.

Winter tick survival, rather than moose density, is probably the major determining factor for outbreaks of severe disease. Adult tick survival is enhanced by warm temperatures, low precipitation and absence of snow cover in April. If, as expected, these conditions are likely to hold over much of the moose range in Ontario, this disease could become more severe and or more common (Leighton 2000a, electronic access).

Adult meningeal worms (*Parelaphostrongylus tenuis*) live as long, thread-like worms in the veins and venous sinuses of the cranial meninges of white-tailed deer. Eggs are passed to the heart and then the lungs, from where they are coughed up, swallowed, and passed into the environment. The larvae are picked up by gastropods (slugs and snails); deer become re-infected when they ingest the gastropods. Survival and speed of development in the gastropods is temperature-dependent. Although the parasites do not seem to cause serious disease in white-tailed deer, they can cause severe neurological illness in some species such as goats, sheep, moose and wapiti. Moose sickness (meningeal worm infection) has been associated with declines in the moose populations of New Brunswick, Nova Scotia, Maine and Minnesota. The impact in Ontario is not well documented.

Warmer summers and lengthening of the frost-free period in autumn, combined with a documented northward extension of the deer range in Ontario and probable increased white-tailed deer populations due to landscape changes, will result in more infections and higher doses of worms per infection. This will have serious, negative health implications for both domestic and wild species sharing the same landscape (Lankester 2001).

5.2. Wild Carnivores and Omnivores

Mustelids (such as skunks, otters, and weasels) can suffer from a giant kidney worm which, although asymptomatic, may cause disease in dogs. Raccoons can carry leptospirosis and rabies. Polar bears (*Ursus maritimus*) are carriers of the parasite *Trichinella nativa* and black bears carry *T. spiralis*. All of these are zoonoses with unknown but probably minimal effects in these wild species.

5.3. Wild Rodents and Lagomorphs

Many wild rodent populations respond to changes in weather (and the climate which the weather reflects) through changes in the available food supply. Rodents help to maintain many infectious zoonoses in nature, such as plague, leptospirosis, Lyme disease, and trichinellosis. In all cases, our concern has generally been with the risk to people, rather than to the rodents themselves. *Yersinia pestis* causes population crashes in ground squirrels in the southwest United States, and could cause similar effects in Ontario. The systemic ecological effects of such crashes have not been well studied.

The zoonotic protozoan *Giardia* has been found to infect a wide variety of animals in Canada, including dogs, cats, birds, and various voles and rats, as well as beavers (*Castor canadensis*) (Olsen and Buret 2001). Infections in most animals are asymptomatic; however, young animals may develop chronic diarrhea.

Tularemia and Q fever may be carried by a variety of rabbits and hares. Again, since these are thought to be primarily of importance as zoonoses, they are discussed more fully in Part B of this report.

5.4. Wild Reptiles and Amphibians

Diseases of Ontario's reptiles and amphibians are not well understood. However, given their close evolutionary and ecological association with local landscapes, they are likely to suffer both direct and indirect damage through increases in UV radiation and changes in temperature variations, average temperatures, and rainfall patterns. Diseases in wildlife often become important only when the animals are under stress for other reasons; they then appear as opportunistic infections (abscesses, pneumonia, septicemia; cryptosporidiosis in snakes). Their fate will thus be shared with the integrity of many of the ecosystems and interacting populations within which they live. As with many wildlife species, changes in population dynamics of reptiles and amphibians will have broad ramifications for other species in these ecosystems.

5.5. Wild Birds

Hailstorms have been reported to cause substantial mortality in waterfowl (Stout and Cornwell 1976). One report describes two extensive hailstorms in Alberta that affected approximately 3000 km^2 of waterfowl habitat and killed 64,120 to 148,630 waterfowl (Smith and Webster 1955). Increases in such events in Ontario, particularly around areas such as Long Point where large numbers of birds congregate during migration, could have catastrophic effects for bird populations on the continent.

North American waterfowl populations are congregating into smaller staging areas because of the decreased availability of wetlands. In the last decade, tens of thousands of birds have been dying of diseases that have not previously been reported in outbreak form, including Newcastle disease, avian cholera and duck plague (Brown 2000).

In 1999, major outbreaks of Type E botulism killed piscivorous birds, including common loons (*Gavia immer*), horned grebes (*Podiceps auritus*) and red-breasted mergansers (*Mergus serrator*), on Lake Huron and Lake Erie (Campbell and Barker 1999). The outbreaks occurred in the fall when fish-eating birds congregate in favourable feeding habitat during fall migration. Very little is known about the ecology of Type E botulism. Outbreaks likely occur only when a specific set of environmental conditions occur. These include: a source of bacterial spores (*Clostridium botulinum* Type E spores are widespread in the aquatic environment) and a suitable nutrient-rich anaerobic substrate for growth of the bacterium and production of toxin (such as dead fish). Outbreaks begin when fish ingest the toxin (or local toxin is produced in the gut of living or moribund fish), and birds eat the fish (Leighton 2000, electronic access). Botulism outbreaks appear to be temporally associated with a recurrence of oxygen-depleted "dead zones" in Lake Erie, which have re-appeared since the late 1990s after decades of apparent improvements in the lake. Climate changes, associated with increased temperatures and lower water levels, appear to be contributing factors (Associated Press, 2002).

5.6. Diseases Affecting Aquatic Wildlife

Rather than focusing on specific diseases in aquatic environments, species by species, it seems more useful to examine the complex systemic interactions in those ecosystems and the effects of climate change on disease dynamics in such systems.

The predicted changes in temperature and precipitation in Ontario will have a serious impact on almost all environmental conditions in aquatic systems. Parasites of aquatic organisms are sensitive to temperature change, not only in terms of direct effects of their life cycles and transmission, but also on host biology. However, the anticipated changes in temperature and precipitation also will indirectly affect the distribution and abundance of parasites and their hosts in aquatic systems. Alterations in water levels, eutrophication, stratification, ice cover, acidification, currents, ultraviolet radiation and other abiotic factors all affect aquatic stages of parasite life cycles and their aquatic ecosystem hosts. Therefore, making correct predictions for complex aquatic ecosystems is very difficult (Marcogliese 2001).

Temperature is the most important abiotic parameter and affects parasites of free-living and farmed fish at all life-cycle stages, as well as the fish themselves. In general, increases in temperature accelerate growth rates, development and maturation in cold-blooded organisms. As a consequence, parasites should be able to complete even complex life cycles more rapidly (Magnuson et al. 1997). In temperate latitudes, fish parasites possessing complex life cycles typically produce one or two generations per year. An earlier onset of spring combined with increased temperatures and longer growing seasons could lead to the production of one or more additional generations annually. Temperature affects release of eggs or larvae by adult worms, embryonic development and hatching, longevity of free-living stages, infectivity to intermediate hosts, development in these hosts, infectivity to definitive hosts, time to maturation, and the longevity and mortality of adults (especially in fish hosts) (Marcogliese 2001). In addition, temperature plays a key role in host feeding and behavior, host range and ecology and host resistance in fish. In fact, seasonal feeding patterns of fishes explain much of the variation in populations of adult cestodes, nematodes and acanthocephalans.

5.6.1. Freshwater Systems

For the Great Lakes, populations of cold-water species such as salmonids and coregonids should experience declines and reduced ranges, whereas warm water species such as cyprionids, esocids, centrachids, and ictalurids will expand their ranges (Meisner et al. 1987). There is also a strong potential for 27 warm-water species of fish to invade different parts of the Great Lakes, potentially bringing with them up to 83 new invading parasite species (Marcogliese 2001). These introduced new parasites may affect immunologically naïve endemic fishes and could cause substantial declines in biodiversity.

The seasonal and spatial distributions of invertebrates and fish are often temperature dependent. The synchronicity of their population dynamics, with resulting predator-prey interactions, is threatened by impending climatic changes. Breakdown in synchronicity in the presence of infected definitive hosts, available intermediate hosts, and parasite reproduction will lead to a disruption of the parasite's life cycle and reduction or extirpation of its population.

The water levels of the Great Lakes and St. Lawrence River are expected to decline. Lower water levels generally imply lower flow rates; these conditions promote retention of free-swimming infective stages, retention of snails (the first intermediate hosts for most digeneans), and development of snail habitat. The prevalence and abundance of various digenean metacercariae, such as *Uvulifer ambloplitis* (black spot disease), *Diplostomum spathaceum* (cataracts), and *Posthodiplostomum minimum* (white grubs) will likely increase if water levels or water volume decline or if currents slow. Other effects of decreased water levels include lost access to spawning grounds as wetlands disappear and lowered water quality.

With increasing temperatures and decreasing water levels, many water bodies in the northeast are expected to become more eutrophic. Eutrophic lakes (those with high nutrient and low oxygen levels) and oligotrophic lakes (those with high oxygen and low nutrient levels) each have characteristic parasite communities: Cyprinid hosts and their parasites are characteristic of eutrophic waters, whereas salmonid hosts and their parasites are more commonly associated with oligotrophic waters. If trophic status changes, host fauna and parasite composition will likely change as well.

Earlier onset and increased stratification intensity in the water column due to increased temperature and decreased ice cover may also alter distribution of hosts, vertical migration of zooplankton, and overlap with fish in the water column.

The duration of ice cover is predicted to decrease in freshwater systems in northeastern North America. As a result, hatching and development of parasites may be accelerated, and birds may remain present during all seasons, creating the potential for parasite transmission throughout the year as in milder coastal climates.

As a result of the predicted increase in precipitation and runoff, headwater streams are expected to suffer increased acidification. Under low pH conditions, the diversity of fish, various intermediate hosts, macroinvertebrates, zooplankton, and parasites decreases. However, if snowmelt is reduced, runoff and thus acidification should be reduced in lakes.

Declines in dissolved organic carbon (DOC) input to small boreal lakes as a result of decreased stream flows, drier soils and lower water tables in wetlands will cause waters to become clearer, allowing deeper penetration of solar energy and associated deepening of thermoclines (Schindler 2001). Declines in DOC in small lakes will also exacerbate the effects of increased UV-B radiation expected from depletion of stratospheric ozone (Schindler 1997). UV-B may cause immunosupression of amphibians and fish; Little and Fabacher (1994) showed that UV-B exposure of the magnitude expected in shallow alpine streams left some trout species susceptible to fungal infections, decreasing their survival.

Climatic warming may act with other human perturbations to change the mercury cycle in freshwaters (Schindler 1997). As surface waters become warmer, the ratio of mercury methylation to demethylation should increase, causing greater contamination of aquatic fauna.

Extreme weather can influence the survival and abundance of fish species in certain habitats. Increased wind velocity coupled with loss of tree cover due to forest fires can expose small lakes to more wind, resulting in a net increase in thermocline depth and a decrease in subthermocline habitats available as summer refuge for cold-water species (Schindler 2001). In turn, parasitic cycles will be altered in those communities.

The most profound impact of climate change, in association with other Great Lakes Basin changes, is likely to be the continued flip-flop of parts of the basin among different configurations of interacting species ("attractors"), each of which involves changes in species makeup and diseases (Regier and Kay 1996).

5.6.2. Marine Environments (Hudson's Bay and James Bay)

In ecosystems with relatively simple structure, such as Arctic food webs, a small disturbance can lead to a cascade of changes. This possibility of radical, unpredictable change is identified as a real danger for Arctic ecosystems (Hansell et al. 1998).

Arctic marshes are very vulnerable to global warming, because most are very flat and because freshwater marsh plants are relatively intolerant of saline conditions. As sea levels rise, saline waters will intrude farther into the marsh, especially during storm surges, damaging existing vegetation, decreasing marsh area over time, and affecting the invertebrate and vertebrate communities. These changes may affect the Hudson Bay lowlands. The IPCC 2001 report states that loss of migratory wildfowl and mammal breeding and forage habitats will occur within the southern Arctic ecozone, which is projected to nearly disappear from mainland areas.

Standard predictions from general circulation models show climate change in the Canadian Arctic will bring higher temperatures in winter, increased precipitation throughout the year, and a longer open water season caused by thinner ice cover, earlier breakup, and later freezeup. Climate warming is not expected to result in large increases in summer water temperature, at least in the short term, because the heat budget for Arctic waters largely involves changes of state rather than changes in the heat capacity of water, and because most of the warming will come in winter (Hansell et al. 1998).

Climate-induced change in the environment affects the health and productivity of marine ecosystems over extended spatial and temporal scales. The current trend toward a warming climate could result in modifications to the basic biological properties of many marine populations, thereby making them more susceptible to disease (Harvell et al. 1999). A long-term warming trend, coupled with extreme ENSO events and human activities that have modified marine communities, appears to be contributing to the emergence of new diseases of marine organisms worldwide (Harvell et al. 1999).

Climate warming is predicted to result in an absolute increase in total marine primary production in Arctic water, caused almost entirely by an increase in phytoplankton, with expected increases in total energy flow and production at higher trophic levels. Marine biodiversity should increase at high latitudes (over the long-term, centuries possibly) in seasonally ice-covered water, assuming, however, that communities and individual taxa are mobile and can move northward. In the case of Hudson Bay, there are only two connections through which marine biological invasions can occur (Fury and Hecla Strait and Hudson Strait). While future warming of the basin may increase the opportunity for north temperate species to invade southern Hudson Bay, they first must migrate as far north as and penetrate Hudson Strait. This complex migration may result in at least a temporary reduction in biodiversity in southern Hudson Bay, as Arctic species recede and temperate species slowly respond.

Another consequence of climatic warming and increased precipitation at high latitudes is that rivers could deposit increased amounts of pollutants into Arctic marine ecosystems. Pollutants are already accumulating in the Arctic, and marine mammals, as long-lived apex consumers with high lipid content, are likely repositories for contaminants. Contaminants can also be assimilated through the ice-edge food web, by fallout of airborne pollutants on sea ice. Programs that monitor the flux and bioaccumulation of pollution in Arctic systems should be integrated with climate change programs examining and modeling oceanic and atmospheric circulation (Tynan and DeMaster 1997).

Effects of climate change on arctic marine mammals include loss of ice-associated habitat; regional or seasonal shifts in prey availability, which can affect nutritional status; changes in reproductive success and geographic range; and alterations in the timing or patterns of migration, which may produce changes in species distributions and stock structure (Tynan and DeMaster 1997). Ice-edge communities, including sympagic ("with ice") algae and crustaceans, and the Arctic cod (*Boreaogadus saida*) which feed on them, are pivotal components in the Arctic food web. Regional decreases in the recruitment of Arctic cod larvae, due to a loss of critical ice-edge habitat or alteration in the seasonal timing of spring blooms, should adversely affect marine mammals (Tynan and DeMaster 1997). Similarly, the recruitment level of capelin (*Mallotus villosus*), an important food species of belugas (*Delphinapterus leucas*) in Hudson Bay, is expected to affect the foraging success and distribution of this and other predator species.

Alterations or loss of existing habitat change population behaviour and species distribution and abundance, which may influence the accompanying distribution of infectious diseases.

A reduction in seasonal ice cover and increased open water should shift the distributions of many vertebrates north. However, colonial seabirds have large established nesting colonies and are slow to move to new sites, so access to water formerly under ice is unlikely to result in rapid range extensions. Those species that feed at marginal ice zones may need to forage at increasing distances from breeding colonies, with potentially profound impacts on breeding productivity and population size. Increased time foraging in warmer climates could be offset by a reduction in chicks' requirement for brooding (Thompson and Hamer 2000). Bearded seal (*Erignathus barbatus*), narwhal (*Monodon monocerus*), beluga whales, and walrus (*Odobensus rosmarus*) are likely to exploit resources in new open water quickly, but their southern range limits could shrink at the same time. Thus, marine mammal and seabird diversity should either decrease or remain the same in Hudson Bay and other low Arctic waters, where Arctic species are replaced by more southerly ones.

The ringed seal (*Phoca hispida*) needs stable snow-covered ice for giving birth, and at the southern end of its range, early snow and ice melt can affect pup survival. Ringed seal distribution should shrink as the southern edges of their range become less suitable habitat. The distribution and abundance of polar bears should follow closely that of its primary prey, ringed seal. Hudson Bay remains a special case because it may become mostly ice-free. If this happens, ringed seal reproduction will decrease sharply, and with it, polar bear populations. At the southern limits of polar bear distribution, these animals are already fasting about four months during the summer when the ice melts. Prolonging the ice-free season will increase nutritional stress and starvation. Monitoring the movements, population status and conditions of polar bears and ringed seals in Hudson Bay could verify the effects of persistent climate change in the region (Tynen and DeMaster 1997).

The effects of reduction in ice cover and extent may affect arctic cetaceans through linkages to prey availability. Beluga whales forage at ice edges and ice cracks. Seasonal shifts in the distribution of bowhead whales (*Balaena mysticetus*) and belugas with ice cover have been observed; however, less is known about the degree of plasticity within species and between stock to adapt to longer-term changes in sea ice extent and prey availability.

Epizootics of disease associated with morbillivirus infection have been recently documented in several species of seals and dolphins around the world (Harvell et al. 1999). Serological studies have shown that morbilliviruses are ubiquitous among cetaceans and are probably transmitted periodically between species (Duignan et al. 1995). Phocine distemper virus was thought to be transmitted to previously unexposed seals of northwestern Europe by infected harp seals, which in the late 1980s migrated toward Europe in response to food shortages due to overfishing around Greenland (Heider-Jorgensen 1992). Climatic change could alter behavior and movement patterns of aquatic mammals, thereby influencing the occurrence of future morbillivirus epizootics in immunologically naïve populations (Kennedy 2001).

6. References

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Dr. Harvey Artsob, Zoonotic Diseases and Special Pathogens, National Microbiology Laboratory, Population and Public Health Branch, Health Canada, Winnipeg, MB. March 2002.

Dr. Cheryl Waldner, Professor, Veterinary Epidemiologist Department of Herd Medicine and Theriogenology, Western College of Veterinary Medicine, University of Saskatchewan, Saskatoon, SK, May 2002.

Dr. Paula Menzies, Department Population Medicine, University of Guelph, Guelph, ON. June 2002.

Dr Richard Ostfeld, Researcher, Institute of Ecosystem Studies, Box AB Route 44A, Millbrook, NY 12545. June 2002.

A Synopsis of Known and Potential Diseases and Parasites of Humans and Animals Associated With Climate Change in Ontario

Part D.

Adaptation and Mitigation Measures

1. Background: Rationale for a Response

As previously stable patterns of climate change in the coming decades, people and animals in Ontario will likely experience different health issues than before. Because the role of climate in determining health outcomes is complex, and because interactions among human health, animal health, and the environment are complex, it is impossible to predict exactly how climate change will affect health. Yet, clearly, a response is needed in Ontario and elsewhere (NRC 2001). A general understanding of existing health-related vulnerabilities to climate, together with output from climate change model scenarios, provides some basis for preparing a response. Because it is based on uncertain information, this response must consist of adaptive strategies that are reviewed and improved as new information becomes available.

Eco-social systems are fundamentally complex, and our human communities are nested within ecological systems that evolved before us and that provide the underlying context for human society (Figure D.1, Boyle et al. 1996). The short-term cause-and-effect relationships that interest us (for example, between mosquitoes and disease) are embedded in the non-linear dynamics of these larger systems. The impacts of climate change on disease summarized in this report occur through various feedback loops and across spatial and conceptual scales in a nested hierarchy (local eco-social systems being part of larger communities and watersheds, which are part of eco-regions and larger political configurations). Indeed, systems researchers have concluded that such complex eco-social systems can be understood only by using multiple perspectives (Casti 1994, Kay et al. 1999).

Therefore, it is not surprising that many of the issues relevant to climate change and public health policy are characterized by epistemological and ethical conflicts, high levels of uncertainty, high stakes, and a sense of urgency that "something be done." No one set of experts or single method of investigation or paradigm can capture "the whole truth" of the system. Indeed, a *normal* science-as-usual approach is largely incapable of adequately dealing with the climate change and health issues we are facing. Jerry Ravetz, director of research consultancy in the United Kingdom, and Silvio Funtowicz, a senior researcher with the Institute for Risk Research for the European Commission, have argued that these kinds of questions require a new approach to science, which they call *post-normal science* (Ravetz 1999, NUSAP 2002, electronic access).

Based on long experience with environmental and public health policy issues, they and others argue that the peer group for inquiry and policy development must be integrated and expanded. The investigative enterprise must be linked more closely to policy goals and must incorporate a variety of knowledge types, both qualitative

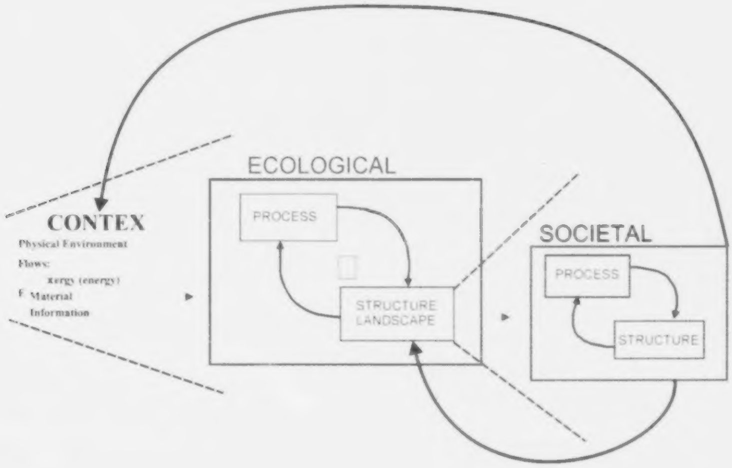


Figure D.1. *A conceptual model (eco-eco model) for nesting human systems in ecosystems (Kay et al. 1999, copyright by NESH www.nesh.ca and Kay et al. 1999).*

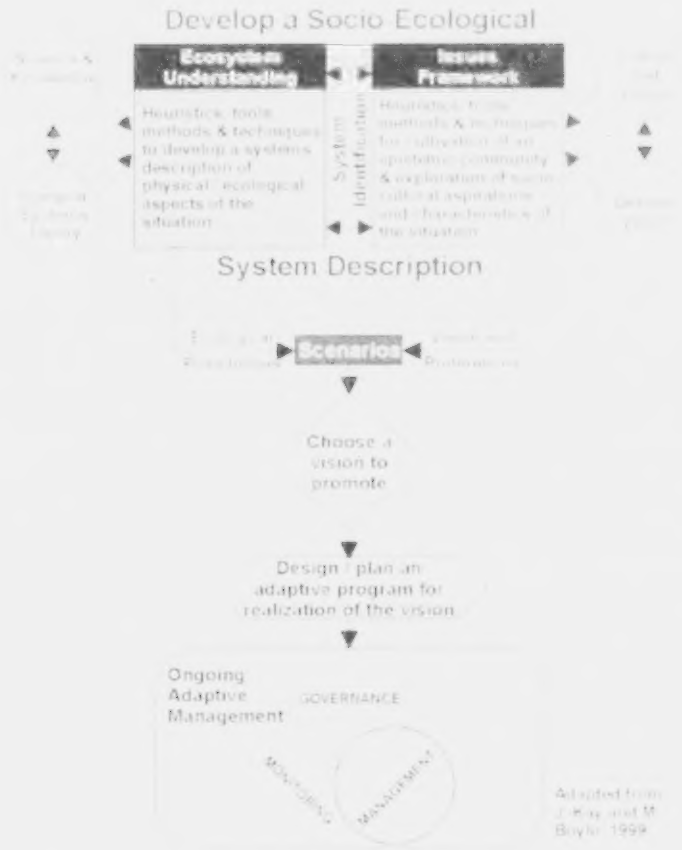
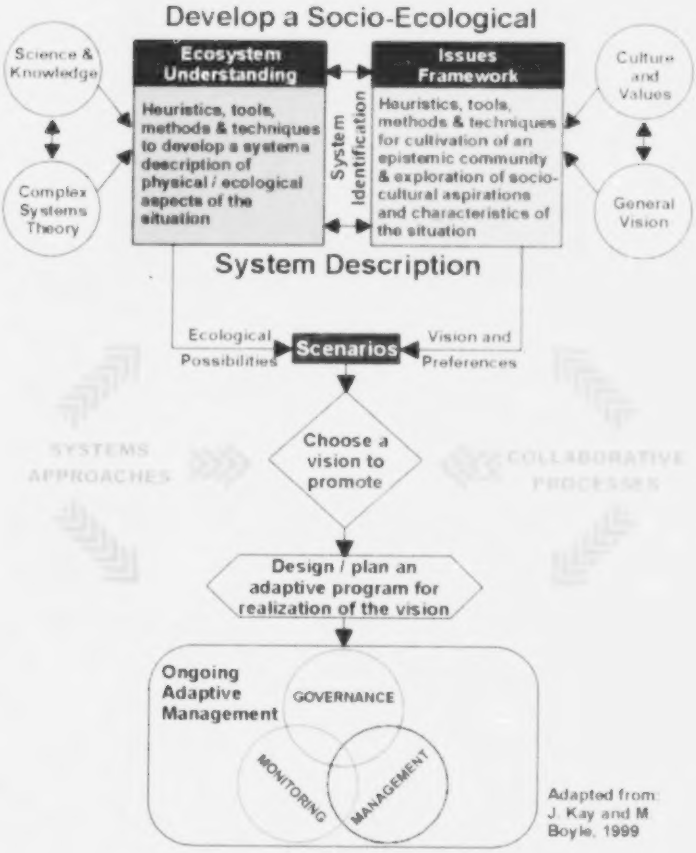

Develop a Socio Ecological
Ecosystem Understanding
Issues Framework
Heuristics, tools, methods & techniques to develop a systems description of physical / ecological aspects of the situation
System Identification
Heuristics, tools, methods & techniques for cultivation of an epistemic community & exploration of socio-cultural aspirations and characteristics of the situation
System Description
Scenarios
Choose a vision to promote
Design / plan an adaptive program for realization of the vision
Ongoing Adaptive Management
GOVERNANCE
MONITORING
MANAGEMENT
Adapted from J. Kay and M. Boyle 1999

Figure D.2. *An adaptive approach to integrating ecological science and social understanding into management (modified from Kay et al. 1999 and NESH 1999 workshop).*

and quantitative, ranging from direct human experience to laboratory experimentation. Their work, among others, has resulted in some novel forms of integration of public participation in the processes previously driven primarily by scientific expertise (Ravetz 1999). Figures D.2 and D.3 are 2 representations of this process. Figure D.2, developed by James Kay and associates at the University of Waterloo (Kay et al. 1999), emphasizes integrating social processes with scientific research to achieve policy goals. Figure D.3 (Waltner-Toews et al. in press) focuses on the iterative nature of problem solving in this context.

Expanding the peer group means, in practice, that we want to involve legitimate stakeholders not just to consult their opinions but also to define questions and modes of inquiry and to judge the quality of the outcomes. This is not describing the world through public opinion, but rather enriching our understanding of the world in a systemic, inclusive fashion. There is no easy way to do this, and there is no obvious institutional framework within which systems scholars, social, natural and biomedical scholars, social activists, business leaders and policy decisionmakers and members of the public can share knowledge and devise feasible solutions. There is no third party to tell us which formulations of the problem or proposed solutions are "correct." We have little choice but to negotiate a consensus based on sets of rules, which have also been negotiated.

In practice, what emerges from the processes of post-normal science is an adaptive environmental management approach first proposed by Carl Walters and C.S. Holling (Walters 1986) but enlarged and enriched to account for the understanding that people are inside complex eco-social systems and that behaving as if we were merely


Develop a Socio-Ecological
Science & Knowledge
Complex Systems Theory
Ecosystem Understanding
Heuristics, tools, methods & techniques to develop a systems description of physical / ecological aspects of the situation
System Identification
Issues Framework
Heuristics, tools, methods & techniques for cultivation of an epistemic community & exploration of socio-cultural aspirations and characteristics of the situation
Culture and Values
General Vision
System Description
Ecological Possibilities
Scenarios
Vision and Preferences
SYSTEMS APPROACHES
COLLABORATIVE PROCESSES
Choose a vision to promote
Design / plan an adaptive program for realization of the vision
Ongoing Adaptive Management
GOVERNANCE
MONITORING
MANAGEMENT

Adapted from: J. Kay and M Boyle, 1999

Figure D.2. *An adaptive approach to integrating ecological issues and social understanding for management (copyright Kay et al. 1999 and NESH 1999 www.nesh.ca).*

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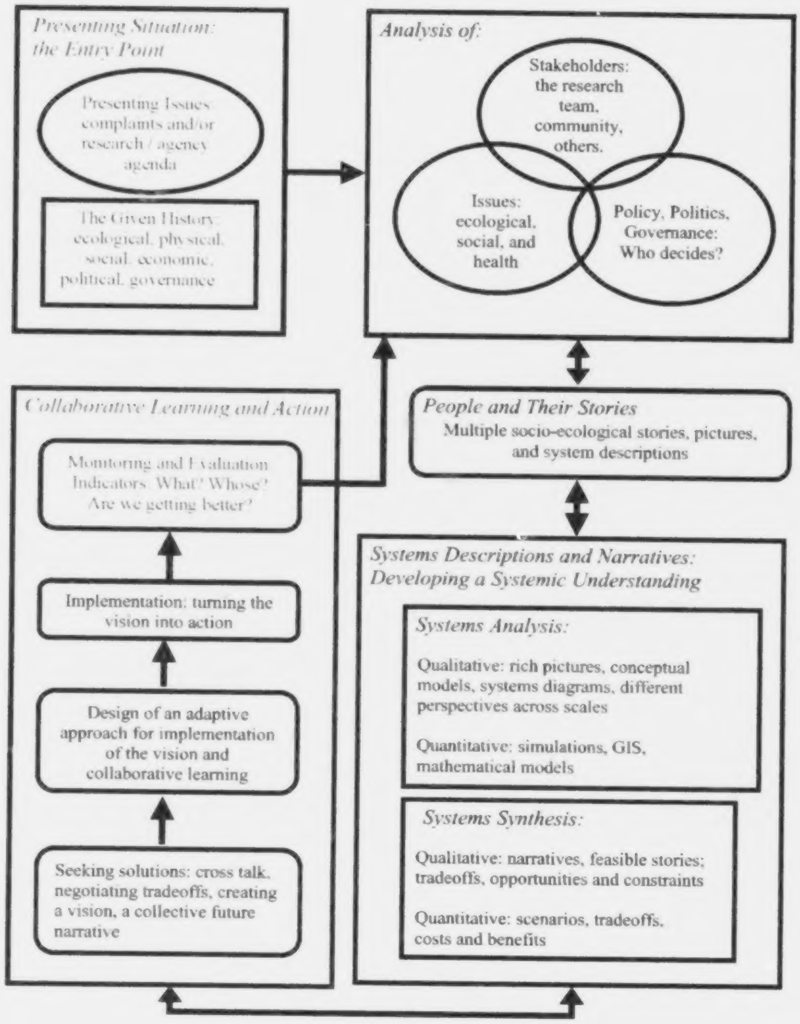
Presenting Situation: the Entry Point
Presenting Issues: complaints and/or research / agency agenda
The Given History: ecological, physical, social, economic, political, governance
Analysis of:
Stakeholders: the research team, community, others.
Issues: ecological, social, and health
Policy, Politics, Governance: Who decides?
People and Their Stories
Multiple socio-ecological stories, pictures, and system descriptions
Systems Descriptions and Narratives: Developing a Systemic Understanding
Systems Analysis:
Qualitative: rich pictures, conceptual models, systems diagrams, different perspectives across scales
Quantitative: simulations, GIS, mathematical models
Systems Synthesis:
Qualitative: narratives, feasible stories; tradeoffs, opportunities and constraints
Quantitative: scenarios, tradeoffs, costs and benefits
Collaborative Learning and Action
Monitoring and Evaluation Indicators: What? Whose? Are we getting better?
Implementation: turning the vision into action
Design of an adaptive approach for implementation of the vision and collaborative learning
Seeking solutions: cross talk, negotiating tradeoffs, creating a vision, a collective future narrative

Figure D.3. *An adaptive methodology for ecosystem sustainability and health (Waltner-Toews et al. 2003, copyright Waltner-Toews et al. 2003 and NESH www.nesh.ca).*

external managers is neither feasible nor desirable. Thus, to respond to the issues raised in this report, we need an organizational arrangement that is flexible, multi-disciplinary, adaptable over time, linking basic scientific understanding with experiential knowledge and policy development. Indeed, the policies themselves must be adaptable. The Health Canada Climate Change and Health Office and Natural Resources Canada's Canadian Climate Research and Adaptations Network (C-CIARN) have made some important steps towards this goal. Research funding from the Climate Change Action Fund and more recently from Health Canada's Health Policy Research Program and the Canadian Institutes of Health Research is providing further impetus in the search for understanding the problems and designing solutions. In making our recommendations, we argue that many activities already begun need to be strengthened and expanded. We believe that they can become prototypes for how such complex issues can be dealt with in general.

2. The Organization of Response

The kinds of organizational structures that appear to hold promise for fostering scholarly inter-disciplinarity, policy relevance, and flexibility are national steering committees comprising researchers and practitioners;

national research and policy networks built around broad themes and incorporating representatives of the public, government and private practitioners, and scientists; and a strong local response capability. These structures need to be set within a national commitment to addressing climate change issues and policies that reflect this commitment. For example, signing the Kyoto Accord, viewed by some to be too small a step toward reducing greenhouse gas emissions, by others as far too costly, and by most as unwieldy, is perhaps the most important indicator of a national and international commitment to address the issues.

Networks and steering committees are already being developed. The Health Canada Climate Change and Health Office has sponsored and facilitated networks on the following climate change impact themes: temperature-related morbidity and mortality, extreme weather events, air pollution-related health effects, water- and foodborne contamination, vectorborne and zoonotic diseases, population vulnerability in communities (including children's health and well-being), and socioeconomic impacts on health and well-being.

These networks need to be strengthened, supported and funded by ministries of health and ministries of the environment at all scales of government. They could link a variety of researchers and practitioners across the country to allow information to be gathered from a variety of sources, reviewed for quality, and redistributed to a wide variety of stakeholders. If they are fully funded, with strong virtual nodes (web pages, full-time managers), they can act as clearinghouses for information that local communities as well as national decisionmakers can draw on to develop appropriate policies and action plans. Networks can also respond to specific issues as they arise, without requiring the critical mass of expertise to reside in any single place. Such networks can draw on expertise from local, provincial and national government departments, as well as universities and other non-government organizations. No other institutional format allows this to happen.

Steering committees, such as the Enteric Disease Surveillance Steering Committee (ENDS)[1], provide a forum for public practitioners and researchers to meet periodically to address specific issues, to identify issues "on the horizon," and to establish strategies for dealing with them. These complement the networks, providing for more direct personal contact and interaction and serving to reinforce the sense of national community in addressing complex environmental and health problems.

At the local level, responsiveness to diseases related to climate change should be integrated with diseases related to the environment in general. Building up eco-health or sustainable development units within public health units, drawing on health practitioners, local businesses, and environmental scientists, would be an effective and cost-efficient strategy. These local units could be tied into national networks for gathering surveillance data (public observations, etc.) and for implementing regional or national programs. The Canadian, provincial, and territorial public health associations, as well as the Council of Chief Medical Officers of Health, have important roles to play in coordinating some of these activities.

3. The Content of Response

If networks and steering committees can provide the organizational framework, what should be the contents? We recommend that human and animal disease networks not be separated, since most of the human diseases of interest are shared in nature with other animals. In some cases, animals such as dogs (*Canis domesticus*) and cats (*Felis domesticus*) share human environments and may serve as early warning indicators of problems. Even when environments are not directly shared, diseases in animals can serve as sentinels for risk areas (for instance, dead crows for WNV), and diseases in people may be linked to wildlife or livestock issues (for instance, waterborne disease outbreaks). Furthermore, we believe that a cross-disciplinary approach will allow for quicker learning and identification of possible technical and engineering adaptations, as well as problems associated with them. For example, draining wetlands may lessen mosquito habitat, but may create serious problems for wildlife and hinder filtering of farm runoff.

3.1. Surveillance and Monitoring

Continuously and routinely recording disease events is the foundation of all health information systems, whether for animal health, public health, or some integrated version of both. For some diseases, well-developed surveillance systems are already in place in Ontario. Although not originally designed with climate change in mind, they can easily be adapted to include surveillance for possible climate-related phenomena.

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[1] This committee is coordinated through the Centre for Infectious Diseases Prevention and Control, Division of Enteric, Foodborne and Waterborne Diseases, Public Health Branch, Health Canada.

The Canadian Food Inspection Agency (CFIA, see list of websites) and the Canadian Animal Health Network (CAHNet, see list of websites) ensure national coordination of monitoring for infectious diseases important for livestock production and trade, as well as zoonotic pathogens in livestock. CAHNet is a national lattice of federal and provincial disease surveillance networks, under the direction of a 16-member committee.

Human health is monitored through a variety of surveillance systems, including Flu-Watch, the National Enteric Surveillance Programme (NESP), and the Canadian Enteric Outbreak Surveillance Centre (CEOSC). Flu-Watch is a federal-provincial collaborative surveillance program that collects information on confirmed cases of influenza and relays it to public health units across the country. NESP is based on the timely national reporting of cases of enteric disease with diagnosis confirmed in provincial laboratories. This system is linked to the international PulseNet system, which allows widespread international sharing of molecular genetic information on laboratory-diagnosed enteric infections. More recently, surveillance of outbreaks of enteric disease has been enhanced by CEOSC. Public health officials linked by electronic list are alerted to suspected or confirmed excess cases of enteric illness detected by local health units. Furthermore, formal and information links are emerging with the World Health Organization and the Pan-American Health Organization.

Animal sentinels are used to assess the risk of rabies in Ontario. With the emergence of raccoon rabies, this animal sentinel surveillance program was combined with a public health intervention: Raccoons (*Procyon lotor*) were vaccinated widely in areas at the front of the spread of the disease. Although this intervention did not prevent the new strain of rabies from entering Ontario, it did slow the spread of the outbreak. It is also an example of the use of geographic benchmarks or targeted surveillance zones. The successful West Nile virus surveillance system is based on monitoring of animal sentinels (mostly crows [*Corvus brachyrhynchos*]) and on vector surveillance (mosquitoes). The system allows public health officials to track the spread of the disease and to measure risk of human exposure. Such coordinated federal and provincial surveillance programs, which involve public health officials working with wildlife and animal health professionals to mount an integrated response to a public health threat, are the type of adaptive system that is needed to address health issues related to climate change. These approaches should be maintained, enhanced and applied to other diseases. Stephen and Ribble (2002) have proposed that outbreaks of diseases in animals could be used as sentinels for emerging environmental health risks.

As has been shown in the case of West Nile virus, effective monitoring can incorporate animal sentinels, an educated public, and a sound diagnostic system. Surveillance may be targeted at specific diseases or focus on more general indicators. Physicians or veterinarians (wildlife, livestock, pet) can be recruited to flag outbreaks, which can be tracked in a central database to identify spatial or temporal trends. Ideally, we should move toward being able to identify vulnerable ecosystems, communities, and populations, as well as individuals (children, the elderly). These surveillance and monitoring systems should be used not just to track diseases but also to conduct ongoing and retrospective research into the dynamics of diseases under different weather conditions. For example, Sutherst (2001) has espoused new modelling technologies (i.e., process-based models) to understand and guide management of parasites under global change. Unfortunately, lack of data for almost all species is the greatest barrier to modelling and other analyses. Clearly, surveillance and monitoring of diseases related to climate change requires coordination across a wide variety of disciplines, professionals and geographic areas.

International coordination of disease surveillance is vital to early detection of emerging disease threats in the coming decades. Because of the importance of Toronto as a travel hub, and of Ontario as a livestock importer, enhanced surveillance of human travel to and from Ontario and of animal movements is particularly important to prevent imported diseases from establishing in a warmer Ontario climate. It could also help protect public and animal health from bioterrorism.

3.2. Public Education and Communication

Educating the people of Ontario about the potential human and animal health impacts of climate change will help them protect themselves and their livestock. Most people take some care in protecting themselves from harmful UV radiation and vaccinate or medicate their pets, horses, and livestock against known diseases. Most people also know to avoid wildlife to protect themselves from rabies. Awareness is increasing about the human and equine health hazards of mosquito bites related to West Nile virus. Similar approaches can be taken regarding tickborne diseases, water contamination following heavy rainstorms or flooding, foodborne illness in hot weather, etc. Providing continuing education to veterinary and public health professionals would also benefit public health, as we rely on these professionals to recognize first cases of new diseases. The better prepared they are, the more likely a new problem will be detected quickly.

To protect the public during extreme weather events, a system of public health alerts and weather warnings can be developed and followed up with pre-specified actions. Two examples of such systems include the widespread broadcasting of a UV index as a measure of severity of damaging solar radiation and the heat-alert system in Toronto. Other types of warning systems might include wellhead and beach contamination alerts linked to precipitation forecasts, alternative power alerts when severe ice storms are anticipated, and ongoing public health warnings about specific diseases (e.g., West Nile virus).

Some sources of information already exist, such as the Health Canada Climate Change and Health site (HC-CCHO, see list of websites). If linked with other sources of information, such as websites associated with networks, the government-based sites can focus on what is within their mandate and be part of the larger network of information. For example, a site known as ECCHO (Ecosystems, Climate Change, and Health Outcomes) is being developed for the networks on foodborne and waterborne diseases, zoonoses and vectorborne diseases. Linked with projects on waterborne diseases and climate change, as well as the Network for Ecosystem Sustainability and Health (NESH, see list of websites), this site will draw on a wider base of information and community-based ecosystem approaches to respond than the government may be comfortable with. These linked sources can serve as a clearinghouse to increase awareness of the risks related to climate-related stress (heat waves, natural disasters, vectorborne diseases, UV rays, disease risk abroad for travellers).

3.3. Intervention

3.3.1. Ecosystem Intervention

Many diseases that may be affected by climate change are embedded in particular ecosystem dynamics; two examples are Lyme Disease (Ostfeld and Keesing 2000a, 2000b) and plague (Enscore et al. 2002, Parmenter et al. 1999, O'Connor 2002, electronic access). Intervening in the ecosystem would appear to be the most straightforward way to prevent some of these diseases, for example, by planting trees, improving water management, and zoning critical watersheds to reduce waterborne disease outbreaks. However, given that ecosystem dynamics are complex (especially in many parts of Canada where wildlife habitat, agriculture and human communities are part of the same ecological mosaic on the landscape) and that local effects are often embedded in larger systems, it is not clear what kinds of interventions would be most appropriate.

Lyme disease and West Nile virus, for example, first made their appearance in Canada in protected wildlife areas and parks. These protected areas (particularly as gathering points on North American migratory bird flyways) are essential for long-term ecosystem integrity, and interventions that would control or eliminate the infections but sustain ecosystem functions are not immediately obvious. Identifying adaptive strategies at various geographic and social scales and the long-term implications of those strategies will require considerable research.

Interventions will need to be structured so that local policymakers can learn from the situation as it changes and evolves. Processes and methods have been developed that integrate research, public participation, policy development, and an understanding of complex systems (Waltner-Toews et al. in press, see figure D.3). As we indicated in the section on organization, there is a need to build local capacity to deal with eco-social dynamics within regional and national strategies. Networks built around local public health and environmental working committees that involve representatives from environmental and health ministries at multiple levels (local, provincial, national) can help foster this effort, both by providing interventions tried by and found useful by some communities and by providing guidance for structuring such interventions within a larger process. The Network for Ecosystem Sustainability (NESH 2003) has focused on international activities in this field, as has the International Development Research Centre's program on Ecosystem Approaches to Health. While both have tended to emphasize organizational approaches, they have recently moved to create partnerships that bring together social learning and scientific knowledge.

In agricultural areas, ecosystem interventions will often appear as management changes. Managing risk related to weather and climate is already an integral part of livestock management in Canada. Many livestock animals are intensively managed under controlled conditions. However, like urban housing and transport strategies, livestock management in Ontario is predicated on a certain, predictable, range of weather conditions. As climate change moves conditions outside that range, resulting in higher average temperatures and more frequent extreme events such as droughts and floods, current management regimes may not be adequate to cope. Barn designs, ventilations systems, and transport between the farm and the food processor will all need to be modified to reflect the changing climate.

Farmers have an array of options for minimizing the negative impacts of thermal stress on production and for exploiting the positive impacts. For livestock, these options include changes in buildings, herd sizes, livestock types or breeds, feeding rations, and heating and cooling systems. Some of these, such as building new barns, probably require too large an investment to be feasible, given the uncertainty of the climate changes ahead. However, many lower-cost systems have already been developed by farmers in warmer climates, and information on these should be made available to Ontario farmers. For instance, protecting animals such as horses against diseases such as West Nile virus will require a combination of vaccination and other management changes, so that horses are not put into situations (near swamps at dusk, for example) where they are at greatest risk. Feedlot managers should plan to change their practices (using sprinklers, altering the timing of handling and/or transport) for managing livestock in very hot and/or humid conditions[2]. The greatest current problem in controlling agricultural parasites is the development of resistance to most available drugs (e.g., sheep in northern Australia) and the pressure to reduce chemical residues in food and in the environment (Sutherst 2001). Resistance continues to emerge because effective resistance management strategies have not been adopted or implemented on a wide scale. The concept of *conservation of susceptibility* needs to be implemented aggressively, as, once susceptibility is lost, it cannot be recovered.

In urban areas, intervening in the ecosystem takes the form of developing infrastructure. Such intervention is discussed in the next section.

3.3.2. Infrastructure Development

When emergencies do occur – heat waves, ice storms, drought – an infrastructure needs to be in place to minimize or limit both disease and its effects. For example, people and their pets will need access to air-conditioning during heat waves and to shelters after other disasters. Livestock will need access to feed regardles of environmental conditions; for instance, during a drought, hay may need to be moved to where it is needed. Equally important are evacuation plans for livestock and people during floods, forest fires, etc., as well as the ability to quickly identify sources of drugs and vaccines and the infrastructure to deliver them. Many of these infrastructure issues relate generally to disaster response capacity and need to be coordinated at multiple government levels.

The infrastructures mentioned above are primarily aimed at emergency response. The kind of infrastructure development needed for long-term mitigation and management could be seen as a kind of ecosystem design and intervention. Reducing carbon emissions by developing and promoting more efficient public transport and discouraging automobile use in cities should be an important public health priority.

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[2] That is, when the THI-based livestock weather safety index reaches alert (75-78, associated with some decrease in the rate of weight gain), danger (79-84, noticeable decrease in weight gain and severe effects if handled, transported or overcrowded), and emergency levels (>84, mortality can occur, especially when heat persists for 3 days or more) (Hahn 1999, Hubbard et al. 1999, Hahn and Mader 1997). (THI is the temperature-humidity index: $THI=T_{db} + 0.36 \times T_{dp} + 41.2$ where T_{db} is the dry bulb temperature in °C and T_{dp} is the dewpoint temperature in °C.)

Steps may be taken immediately to help mitigate some of the potential human health hazards associated with climate change. Some of these involve simple behaviour changes and are easily implemented now. Other steps involve large-scale changes in how we relate to the ecosystems that support us.

Heat-related illness and death are largely preventable through human behavioural adaptations, such as use of air conditioners (provided utilities can deliver sufficient energy to users at those peak times) and increased intake of fluids. Other adaptive measures include developing community-wide heat emergency plans, improving heat warning systems, and producing better heat-related illness management plans (Patz et al. 2000). Smoyer et al. (2000) also recommended policies that promote wise land use to minimize the effects of urban heat islands, provide vegetated and ventilated areas to facilitate cooling, and ensure that health and social services are readily available to populations in need. Other adaptations include increasing urban green space, from roof-top gardens to parks and trees (provided sufficient water is available), and considering mosquito and tick habitat and hazards from vectorborne diseases.

Mitigating public health threats from foodborne and waterborne diseases requires a wider view of food and water safety. Implementing and enforcing watershed management plans, environmental farm plans, and livestock management guidelines can help to reduce pathogens at the source. Integrating public health programs with livestock and wildlife health surveillance would enhance existing systems. Improving the amount and types of antimicrobials used in livestock (*wise-use* systems) will help reduce the spread of antimicrobial resistance, particularly if physicians and veterinarians prescribe fewer antibiotics.

Other zoonotic and vectorborne diseases are less amenable to control by ecosystem level interventions. For example, wetlands provide important mosquito breeding habitat. Yet to eliminate these would disrupt vital parts of aquatic ecosystems, eliminate crucial migratory bird halts, and reduce biodiversity. Similarly, the Carolinian forest ecosystem so favored by the Lyme disease tick vector, the legged tick (*Ixodes scapularis*), is itself threatened and in need of protection and rehabilitation. Widespread spraying of mosquito larvicide has grave consequences for other aspects of the system. Therefore, using vaccines and behavioural change rather than modifying the ecosystem will better address the problems of such vectorborne diseases.

3.3.3. Research and Technology Development

Developing clean energy generation is perhaps the greatest adaptation we can hope for. Much of the technology and engineering necessary to mitigate human-caused climate change is already being developed (clean fuel, low-emission vehicles, UV resistant crops, efficient housing and barn design). However, we need to do more work on understanding the dynamics of the infectious agents in nature and on developing adaptable, cost-effective strategies that will have both immediate benefits (reducing air pollution by reducing car use) and long term climate benefits (reducing human-caused global warming). Is the best response to West Nile virus or Lyme disease simply providing public advisories about when to avoid certain locations and what clothing is appropriate, or is there a role for more dramatic interventions (spraying)? To date, the short-term effectiveness and long-term consequences have not received the research attention they deserve. More research needs to be done to identify community and watershed characteristics that make some areas vulnerable to waterborne disease outbreaks while other communities, similarly stressed, appear to have avoided these problems. Some technologies, or some ways of using technologies, may be more effective than other methods. In addition, some forms of community organization or response may be more effective than others, reflecting more sustainable communities. The research in this area is just beginning and needs to be expanded, with physicians, veterinarians, and public and health environmental researchers on board, as well as significanlty more social scientists.

Our understanding of the health impacts of climate change is still limited. One key research priority in the short term is developing more practical regional climate change projections, which will reqire more highly refined modelling and better (more expensive) computer resources. The more acurate climate chage projections are, the more accurately we can forecast health effects. In some cases, too little is yet known about the impact of climate change on a disease to determine what priority it should have. However, we clearly need to understand the implications of climate change on risks from foodborne, waterborne, and vectorborne diseases. This research requires multi-disciplinary teams of researchers. The researchers and their projects should be linked into research and other networks, for example the climate change health issues networks coordinated by Health Canada and Natural Resources Canada through C-CAIRN. Local public health units should capitalize on the work of such networks to gain access to the latest research news. Furthermore, the participation of local public health professionals in these networks helps provide researchers with questions that need answering. In general, researchers need to more actively inform, educate and work with decisionmakers at all levels of government.

Municipal disaster relief plans for climate-related emergencies should consider a broad range of health issues. Antiquated water supply and treatment put many Ontarians at risk of severe illness. These systems need to be upgraded in anticipation of climate changes, rather than based on historical climate patterns.

3.3.4. Medical and Veterinary Interventions

Medical interventions will include developing new and more effective vaccines and drugs and increasing health-care staff during heat and cold waves, pollution episodes and natural disasters. Successful examples include the Flu-Watch and influenza-vaccination program in Ontario and the rapid Canada-wide distribution and application of equine vaccines for West Nile virus. Although not all impacts of climate change will have the dramatic impact of West Nile virus, quick and effective information exchanges and cooperative action among veterinarians, wildlife biologists, entomologists and medical personnel will provide a coordinated response to the health impacts of climate change. Again, networks (perhaps subsets of those already being developed primarily for surveillance and research) may be the most effective approach. To facilitate the transition of public and veterinary health surveillance teams from surveillance to action-response, additional funding and staff will be required.

3.3.5. Policies and Interventions

To mitigate the effects of, and adapt to climate change, we need a supportive and encouraging policy environment. In general, the most adaptable and effective policies will be focused regionally, but set within a supportive national and provincial/territorial context. Signing the Kyoto Accord will indicate a national commitment, but follow-through policies and actions are also important now. For example, actively supporting networks; making appropriate changes in watershed management, urban housing and transport development; and setting national research priorities are all important. Policy initiatives linking environmental, climate and health issues will require coordination across government departments as well as across scales of governance. Health Canada is working with the World Health Organization and other agencies to develop climate change health impact assessment guidelines. This work must be accompanied by funding for local networks so that communities can develop responses that reflect their local conditions, but can also learn from each other. Communities with unique links to local ecosystems (for example, First Nations) will require special attention, as will new immigrants who may not have a good understanding of the local eco-social context.

The global climate is changing, and this is changing not only the short-term weather patterns that Ontarians are used to but also the human and animal diseases we must prevent or manage. Furthermore, the changing climate is changing the way we manage existing disease issues. We can identify diseases that are likely to be affected by climate and which human or animal groups are most vulnerable to them, but we are unlikely to be able to predict the exact nature of the changes. Improving our knowledge base and our response capability at local, regional and national levels is important to maintaining our current levels of health. This strategy will also enable us to face the coming challenges, adapt to them, learn from them, and continue to create sustainable and healthy communities in this province, in Canada, and internationally.

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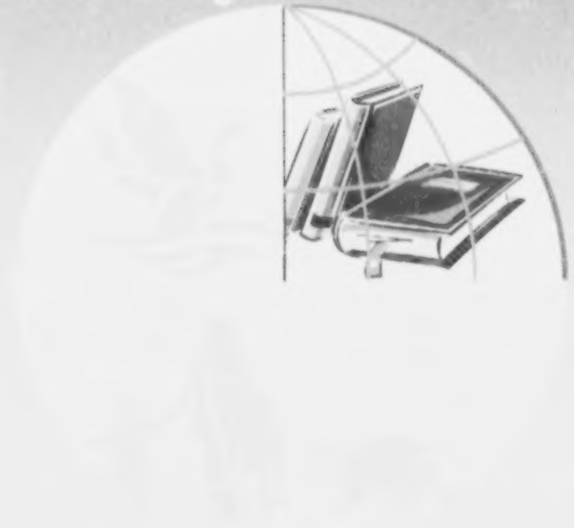
Network for Ecosystem Sustainability and Health (NESH). http://www.nesh.ca

A Synopsis of Known and Potential Diseases and Parasites of Humans and Animals Associated With Climate Change in Ontario



Part E.

Appendices

Appendix 1. Diseases Affected by Ultraviolet Radiation

Stratospheric ozone destruction is a separate process from greenhouse gas accumulation in the lower atmosphere. Stratospheric ozone shields the Earth's surface from incoming solar ultraviolet radiation (UVR), which has harmful effects on human and animal health. In the 1980s, depletion of stratospheric ozone was linked to chlorofluorohydrocarbon (CFC) emissions. Reducing the use of these gases worldwide has reduced the rate of stratospheric ozone depletion. Still, long-term decreases in summertime ozone over New Zealand have been associated with significant increases in ground-level UVR, particularly in the DNA-damaging waveband (McKenzie et al. 1999). Although climate change will not directly contribute to stratospheric ozone depletion, several of the man-made greenhouse gases (e.g., chlorofluorocarbons and nitrous oxide) also deplete the ozone layer. In addition, tropospheric warming (from greenhouse gas accumulation) apparently induces stratospheric cooling, which exacerbates ozone depletion (Shindell et al. 1998, Kirk-Davidoff et al. 1999). Climate change is also expected to alter cloud cover; in some places increased cover may mitigate damaging UVR, and in other places, fewer clouds may result in more UVR reaching the planet's surface. It seems unlikely that the 1-2% increased cloud cover projected for Ontario (Smith et al. 1998) will significantly affect human exposure to UVR. In a warmer world, patterns of personal exposure to solar radiation (e.g., sunbathing in temperate climates) are also likely to change.

Studies show that solar radiation causes skin cancer (melanoma and other types) in fair-skinned humans (IARC 1992, WHO 1994). The most recent assessment by UNEP (1998) projects significant increases in skin cancer incidence worldwide as a result of stratospheric ozone depletion. The role of UV-B in cataract formation is complex: Some cataract subtypes appear to be associated with UVR exposure, whereas others do not.

In cattle, ocular squamous cell carcinoma is one of the most common neoplasms, particularly in older animals (> 5 years) of the beef breeds (Hereford, Simmental) (Radostits et al. 2000). A combination of genetic predisposition (heritable lack of circumocular and corneoscleral pigmentation) and exposure to a carcinogenic agent (the ultraviolet component of sunlight) increases the probability of lesion development. There is a significant association between increasing levels of solar radiation and risk of developing ocular squamous cell carcinoma in cattle. An association between solar radiation and skin neoplasia has also been suggested for some forms of cutaneous hemangioma/hemangiosarcoma in dogs (Hargis et al. 1992) and for squamous cell carcinomas in both dogs and cats (Madewell et al. 1981).

In humans and experimental animals, UVR can cause local and whole-body immune suppression (UNEP 1998). Ambient doses of UVR reduce cellular immunity (Garssen et al. 1998). UVR-induced immune suppression may increase susceptibility to infection, and thus could alter the distribution of infectious diseases. If these changes occur and are combined with increased exposure to infectious agents due to better vector/agent survival or increased population densities, the levels of disease could increase more than they would from any single stressor.

Given the rarity of these diseases in general, susceptible animals could be used as sentinels for high-risk environments or activities.

Appendix 2. Effects of Climate Change on Disease Vector Ecology

1. Direct Effects of Climate on the Abundance and Distribution of Vectors

Increased temperatures accelerate vector metabolic rates, increase biting rates and feeding frequency, and lead to enhanced egg production and increases in population size. The daily survival rate of individual vectors may decrease as temperature rises and an upper limit exists beyond which high temperature is extremely detrimental. Temperature may affect the geographical range and distribution (in terms of latitude and altitude) of vectors since they tend to be limited by minimum and maximum temperature (and humidity). For a given vector, a 1°C rise in temperature has been estimated to correspond to a 90km change in latitude and a 150m increase in altitude in its range. For example, the mosquito *Aedes albopictus*, a competent vector of yellow fever and dengue viruses, was introduced into the United States from Japan in 1985. The species now has extended as far north as Nebraska and Iowa and if temperatures increase as expected, is likely to extend into the populated areas of the eastern United States and southern Canada (Mellor and Leake 2000).

High relative humidity favours most metabolic processes in vectors. At higher temperatures, high humidity tends to prolong survival although increased susceptibility to fungal and bacterial pathogens may offset this advantage. Low humidity decreases in the daily survival rates of many arthropod vectors because of dehydration, but in some cases it may also increase the blood-feeding rate to compensate for the high levels of water loss.

Rainfall limits the presence, absence, size and persistence of breeding sites for most blood-feeding groups of insects, including blackflies (Simuliidae), biting midges (Culicoides), horseflies (Tabanidae) and mosquitoes (Culicidae), with aquatic or semi-aquatic larval and pupal stages. Very heavy or prolonged rain may disrupt vector breeding sites and wash away the immature stages or kill them directly.

Wind speed affects activity levels of biting midges and other small biting flies. Prevailing winds and wind speed significantly affect vector distribution due to passive dispersal. Some insect vectors, including species of mosquitoes, blackflies, sandflies and biting midges, can be dispersed in this manner for hundreds of kilometres. In winds at speeds of 10-40 km/hr, at heights up to 1.5 km and at temperatures between 12 and 35°C, biting midges may be carried as aerial plankton for up to 700 km (Sellers 1992). Most mosquito species are not as susceptible to wind transport, generally staying within a few kilometres of their hatching site.

Increased frequencies of convective storms could affect airborne disease transmission. Airborne dispersal of arthropod disease vectors has been implicated in outbreaks of viral infection and disease. These include western equine encephalitis and eastern equine encephalitis in horses in Canada and the United States (Sellers 1989, Sellers and Maarouf 1988, 1990, 1993), and bluetongue virus and epizootic hemorrhagic disease virus in the Okanagan Valley of British Columbia (Sellers and Maarouf 1991).

Airborne viral transport was implicated in the 1988 outbreaks of Aujesky's disease in pigs in Denmark, possibly due to airborne transport of the pseudorabies virus from Germany (Christensen et al. 1993). Airborne spread of foot and mouth disease (FMD) has been investigated and modelled, ranging from local travel over land (10 to >100 km), to rare airborne transport for longer distances over the ocean, for example, from France to Britain (Gloster 1982, Donaldson et al. 1982, Gloster et al. 1982). Although these diseases are exotic to Canada, pseudorabies still occurs in domestic and feral swine in some American states and FMD virus may have spread via the air during the last Canadian outbreak in Saskatchewan in 1951 (Daggupaty and Sellers 1990). If the disease enters Canada again, climate and weather variables will complicate control or eradication.

2. Indirect Effects Via Environmental Change

One vector species may be displaced by others in response to environmental changes such as deforestation, expansion of irrigation, or increases in brackish water breeding sites due to a rise in sea level. If a vector were to be accidentally introduced into an area through human activity, climate change may alter its chances of establishing breeding populations.

When attempting to predict how climate change will affect the distribution of vectorborne diseases in Ontario, temperature-related vector and pathogen development rates must be considered simultaneously. In many cases, the successful completion of the developmental cycle of a pathogen within the vector may occur only within a clearly defined temperature band. Therefore, for each vector-pathogen combination, there exists a band of permissive temperatures that is unique to each pathogen and outside of which the pathogen will invariably fail to be transmitted, although the vector itself may be able to survive.

3. Effect on Vector-Viral Biology

Climate change may affect vector competence. Within vectors, rates of viral infection, virogenesis and transmission are temperature dependent. At very high temperatures, better viral survival and transmission would likely be offset by shortened individual vector survival. Northerly extensions of vector range would bring viruses in contact with new potential vector species. Climate change may affect the competence of non-vector species in transmitting pathogens. For example, temperature increases may convert less efficient vector species into more important vectors. Increased temperatures during development may increase the oral susceptibility rate of previously marginal vector species, possibly through giving rise to smaller, more fragile adults with an increased incidence of the so-called "leaky gut" phenomenon. In this phenomenon, the virus passes directly from the blood meal through the gut into the haemocoel, without having to first infect and replicate in the gut wall cells. Once in the haemocoel, most arboviruses will replicate and may be transmitted even by insects that do not normally act as vectors.

Some strains of insects can hibernate during cold weather, providing a viral overwintering mechanism without vertebrate involvement. In temperate areas, many arboviruses are likely to be maintained over the cold winter periods by vector transovarian transmission into resistant eggs.

4. Effects on Macro Parasite Ecology (Abundance, Distribution, and Changing Life Cycles)

Ambient temperature affects parasite life cycles, transmission, and in some instances host biology. Many stages of the parasite life cycle may be affected, particularly during free-living periods or if the parasite cycles through poikilothermic hosts whose development may also be temperature dependent.

Indirect climatic effects may include geographic changes in host distribution and abundance. For parasites that require multiple host species, an altered synchronicity of population dynamics of parasites and hosts together with changes in host distribution may provide an impetus or selection pressure for host switching and alterations or extensions of host specificity. With respect to stochastic environmental effects, extreme weather can influence the survival and abundance of vectors transmitting disease. Stochastic extreme events may result in catastrophic responses, ranging from local extinction to epizootic outbreaks of parasites. Climate-mediated stress may compromise host resistance and increase the occurrence of opportunistic diseases through shifts in the distribution of either hosts or pathogens.

Potential impacts of global warming on arthropods and disease vectors include accelerated development rates and longer growing seasons that change seasonal phenology and abundance and increase the number of generations each year. Parasites may appear earlier in the season, and each subsequent generation would appear earlier in the year. The greatest proportional effects will be evident in populations of species that have few generations each year, such as one-host ticks. Shorter and milder winters will increase survival of parasites through winter, especially for subtropical species. Intensified hydrological cycles caused by increased temperatures (resulting in fewer but heavier rainfall events and higher evaporation rates) will lead to greater extremes of wetness and dryness, and more variable numbers of parasites. Higher temperatures could result in reduced protein content in pastures and greater nutritional stress of livestock, thus reducing resistance to parasites. Higher CO_2 concentrations may increase biomass in plants, which may provide more shelter for free-living stages of parasites and provide a favourable microclimate during more of the year, leading to higher survival for free-living stages (Sutherst 2001).

Climate change may influence the distribution and abundance of various parasitic diseases of wild animals, particularly those with free-living stages or invertebrate intermediate hosts. Whether the range of these diseases shift, expand, or contract depends on individual life cycle components of each parasite group and range shifts/expansions in both definitive and intermediate hosts. Selected examples are included in Table E.1.

Dobson and Carper (1992) postulate that in temperate regions, increasing temperatures and desiccation could limit the distribution of some parasites. Epidemiological patterns would be altered, possibly leading to reduced levels of parasitism or to wider dissemination of species tolerant of higher ambient temperatures. Alternatively, the response of hosts to pathogens could also be modified. For example:

- Increased stress can change host susceptibility to parasitism and parasite-induced mortality.
- Changes in host and parasite distribution can lead to overlapping ranges and increased host-switching.
- Some pathogens could be disseminated more broadly.

In the Arctic and Subarctic, however, changes may be particularly pronounced and radically different from those in boreal regions. At high latitudes, climatological changes and impacts may lead to:

Table E.1. *Effects of climate on vector and disease ecology (from Mellor and Leake 2000, Mellor et al. 2000).*

Temperature	• Increased temperature accelerates vector metabolic rate, biting rates and feeding frequency; leads to enhanced egg production and increased population size. • Daily survival rate of vectors decreases as temperature rises; an upper limit exists beyond which temperature is detrimental. • Temperature affects geographical distribution of vectors; a 1°C rise in temperature is estimated to correspond to 90 km increase in acceptable latitude and 150 m increase in acceptable altitude for a specific vector. • Within vectors, rates of viral infection, virogenesis and transmission are temperature dependent (balanced by shortened individual vector survival at very high temperatures). • Increased environmental temperature converts less-efficient vector species into more important vectors.
Humidity	• High relative humidity favours most metabolic processes in vectors; at higher temperatures, high humidity prolongs survival, although increased susceptibility to fungal and bacterial pathogens may offset this advantage. • Low humidity decreases daily survival of many arthropod vectors because of dehydration; in some cases also increases blood-feeding rate, an attempt to compensate for high levels of water loss.
Rainfall	• Rainfall limits presence, size and persistence of breeding sites for most blood-feeding insects, including mosquitoes, with aquatic or semi-aquatic larval and pupal stages. • Very heavy or prolonged rain may disrupt vector breeding sites and wash away immature stages or kill them directly.
Wind	• Prevailing winds and wind speed affect passive dispersal levels of vectors; some insect vectors disperse for hundreds of kilometres.
Environmental Change	• One vector species may be displaced by another; new host populations may be exposed. • If a vector is accidentally introduced into an area, a suitable climate enhances chances of establishing breeding populations. • Changes to vector range may bring viruses in contact with new potential vector species.

- Latitudinal shifts in geographic ranges
- Extension of the growth season with earlier springs and a broadened window for transmission
- Reduction in developmental times linked to higher temperatures
- Decrease in generation times
- Increase in rates of transmission, larval survival and availability
- Increases in the prevalence and intensity of some parasites

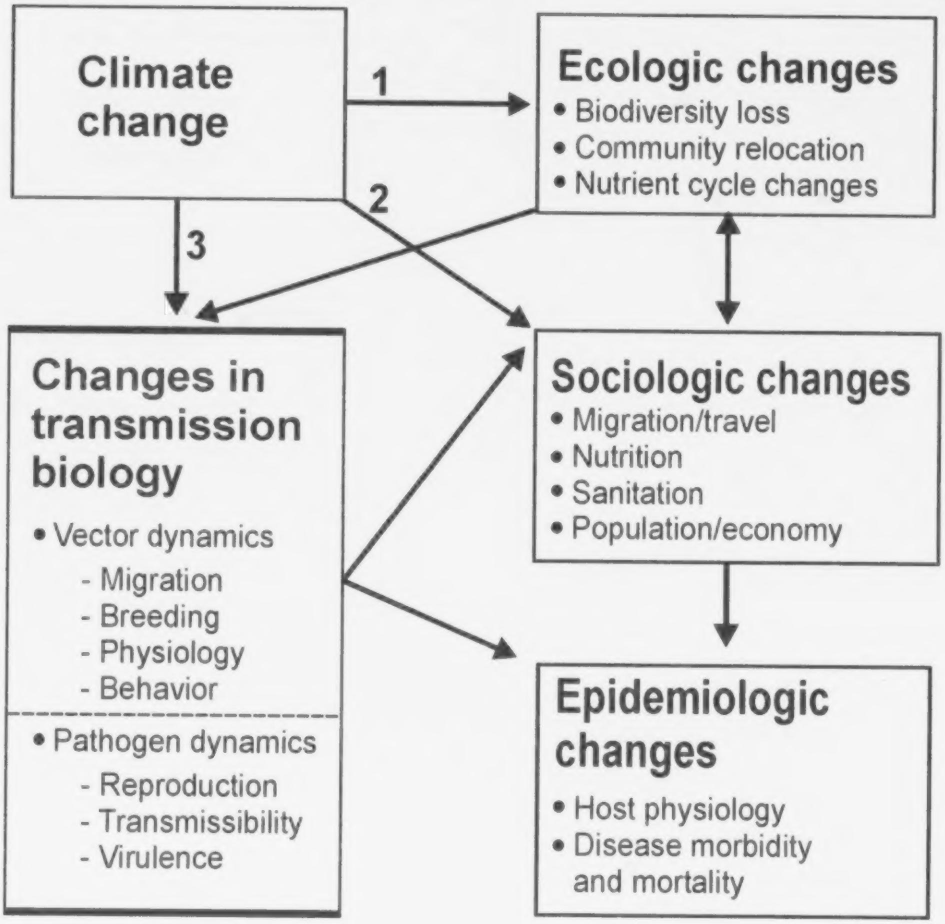
In Arctic host-parasite systems, the potential for parasite populations to amplify and sub-clinical or clinical effects to emerge depends on the degree to which ambient environmental and ecological conditions limit parasite abundance (Hoberg et al. 2001).

General effects of increased temperature on parasites include rapid growth and maturation, earlier maturation in the spring, increased mortality, increased number of generations per year, earlier transmission, and potential maintenance of transmission year-round. The duration of low winter temperatures, rather than the minimum temperature, is most important for maturation, mortality and re-infection. Thus, duration of the growing season becomes critical for parasite population dynamics. Furthermore, the more complex the parasite life cycle, the more likely it is that the parasite will be influenced by environmental parameters (Overstreet 1993).

Since the distribution of a multi-host parasite is dependent on that of all hosts in its life cycle, a modified host distribution will determine where parasites persist and where they colonize. Climate change will influence the distributions of free-living fauna, including fish and aquatic invertebrates, which will affect how their parasites are distributed.

Computer software for predicting the potential distribution and relative abundance of species in relation to climate has been developed (CLIMEX 2002, electronic access). This program, developed by CSIRO Entomology in 1985, has been used to examine the distribution of insects, plants, pathogens and vertebrates for a variety of purposes, including impacts of changes in climate and climate variability.

Appendix 3. A Framework for Assessing Potential Impacts of Climate Change on Vectorborne Diseases *(From Gubler et al. 2001)*


Climate change
1
2
3
Ecologic changes
• Biodiversity loss
• Community relocation
• Nutrient cycle changes
Changes in transmission biology
• Vector dynamics
- Migration
- Breeding
- Physiology
- Behavior
• Pathogen dynamics
- Reproduction
- Transmissibility
- Virulence
Sociologic changes
• Migration/travel
• Nutrition
• Sanitation
• Population/economy
Epidemiologic changes
• Host physiology
• Disease morbidity and mortality

Arrows 1, 2, and 3 indicate direct climate change effects.

Appendix 4. Record of Consultations

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Climate Change and Plant Disease in Ontario

Climate Change and Plant Disease in Ontario

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Executive Summary

Current climate change models for Ontario predict that mean annual temperature will increase by up to 3°C over the next century. Temperatures are expected to increase more in winter than in summer, with an increase in frost-free days, and to increase more in northern than southern latitudes. Minimum temperatures should increase more than maximum temperatures. Precipitation, although somewhat unpredictable, is expected to increase in Ontario, but increased evaporation and transpiration due to warmer temperatures will likely result in drier conditions, particularly during the summer growing seasons.

These anticipated changes will affect the occurrence of both biotic and abiotic plant diseases in agriculture and forestry. In agriculture, climate change is expected to have direct, multiple effects on the epidemiology of plant diseases, including such stages as the survival of primary inoculum of biotic plant pathogens, the rate of disease progress during a growing season, and the duration of epidemics. These effects will influence individual pathogens in a positive or negative manner but, in general, there will probably be more change in the types of plant diseases that develop than in their average annual severity. Changes in the spectra of plant diseases that are economically important are also anticipated in forestry.

Abiotic diseases associated with environmental extremes, such as drought, flooding, wind damage, and hail, are expected to increase in both agriculture and forestry due to the increase in more extreme weather events associated with climate change. These changes will particularly impact crop plants and trees grown on marginal lands, where even slight changes in environmental conditions may stress plants beyond recovery.

Interactions that occur between biotic and abiotic diseases may represent the most important effects of climate change on plant diseases in Ontario and elsewhere. Interactions of biotic and abiotic factors can cause susceptible species to deteriorate gradually, often resulting in plant death. Such diseases are referred to as *decline* diseases and primarily affect forest tree species but can also affect other perennial woody species in agriculture and horticulture.

Management of plant diseases will also be affected by anticipated changes in climate. The research priorities of plant breeding programs may have to adapt to increased duration of growing seasons and to focus on developing drought and stress tolerance in field and horticultural crops. Irrigation use may increase in Ontario to provide adequate moisture during drought periods, requiring increased infrastructure and expertise. Some drought-prone areas used for commercial agriculture and forestry may be abandoned. Changes in climate may allow new crops and cultivars to be introduced and grown in Ontario, but effective systems must be in place to prevent new pathogens from accompanying these new crops. Significant changes may also occur in forestry. Due to the long-lived nature of trees, forests are slow to adapt, and forest management plans will need to consider the impact of climate change.

For over 100 years, agriculture and forestry have been adapting to environmental changes in Ontario. These adaptations may need to occur at an accelerated rate because of the expected rapid changes in climate. The infrastructure of agricultural and forestry research must remain strong to ensure a successful transition and adaptation as Ontario's climate changes.

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Introduction

The potential consequences of climate change have been receiving increased attention in recent years because of growing concern over the effects of higher temperatures due to increasing carbon dioxide (CO_2) levels in the atmosphere. In association with this warming trend, precipitation patterns are expected to change and extreme weather events to increase. These extreme events include severe drought, high and rapidly fluctuating temperatures, ice storms, and severe wind, rain and hail storms. This report will focus on the potential impact of anticipated climate changes on plant diseases in Ontario.

Climatic Assumptions for Ontario

For the purposes of this report, Ontario's climate changes are expected to include an increase in the mean annual temperature of 3°C over the next century. Temperatures are expected to increase more in winter than in summer, likely increasing the number of frost-free days, and will increase more in northern than in southern latitudes. Minimum temperatures will increase more than maximum temperatures. Precipitation, though somewhat unpredictable, is expected to increase in Ontario. However, increased evapotranspiration due to warmer temperatures will result in drier conditions during the growing season. More extreme weather events, with strong winds, heavy rain, and hail, are expected to accompany these climate changes.

Potential Effects of Climate Change on Agriculture and Forestry

Plant health is and should be a national priority of Canada because of our economic and environmental reliance on agriculture and forestry. In Ontario, plant agriculture and forestry are major sectors of the economy; however, little attention has been given to the effects of climate change on plant health.

The amount of land devoted to agriculture, most of which is located in southern Ontario, is approximately 6 million ha and produces crops with a gross annual value of more than $9 billion. Smit and Brklacich (1992) studied the implications of global warming for agriculture in Ontario and provided crude estimates of the sensitivity of agriculture to climate change, based on a small set of global climate model (GCM) outputs. The effects of increased CO_2 concentration on crop yield were not considered. They found that in Ontario, the frost-free season would be extended, growing season mean temperatures would increase, and precipitation over the growing period would also increase because of the longer growing season. However, increases in potential evapotranspiration would more than offset increases in rainfall, resulting in a climate characterized by a longer, warmer, drier, growing season.

Overall, based on these altered climate regimes, Smit and Brklacich (1992) found that crop prospects in northern regions would be enhanced, but the increased area of cultivation would be relatively small. In southern Ontario, moisture stress would reduce yields for many field crops. Land considered prime because of good drainage would produce decreased yields. Provincial production would increase for fodder corn, remain unchanged for barley, and decrease for oats, wheat, and soybeans. While the area of land economically unsuitable for grain corn would decrease, so too would the area for which yields would generate a high return. A larger area of the province, mainly in southwestern Ontario, would have more modest yields (Smit and Brklacich 1992).

An altered climate suggests new opportunities for crop production in northern Ontario, but it is doubtful that this enhanced potential could offset possible declines in southwestern Ontario. An altered thermal regime coupled with relatively low precipitation levels would imply a less favourable environment for agriculture in southern Ontario. In northern Ontario, the benefits of a longer, warmer growing season would also be impaired during relatively dry years (Brklacich and Smit 1992).

Agriculture is well adapted to typical or average growing season conditions but is susceptible to extreme conditions. Changes in mean annual temperature of one or two degrees are rarely considered a problem. However, changes in mean conditions will result in more frequent extreme weather events, even without any change in the variability of climatic conditions. The frequency of extreme events can be greatly changed with even very small changes in the value of the mean. A problematic condition (e.g., severe drought) that currently has a probability of occurring every 30 years may begin to occur every 4 or 5 years (Smit 1999). Changes in long-term climatic variability, or simply shifts in extremes because of changing averages, may well have greater impact than changes in climatic averages (Brklacich and Smit 1992).

In Ontario, crop yields are significantly impacted by diseases every year. The effects of climate change on crop health and particularly on crop disease are not known. Crops may benefit from warmer temperatures and elevated levels of CO_2 in the atmosphere but may be stressed by drier soils and extreme weather. Stressed plants are more susceptible to disease. In addition, pathogens that cause crop diseases will be directly influenced by changes in climate, positively or negatively, depending on the environmental conditions they require to cause disease.

Because of the long-lived nature of forests, climate change that occurs over a relatively short period of time can have serious consequences for forest health. Ontario has over 1 million ha of forest, which makes up 74% of the land cover. This forested land includes 3 major forest regions, the deciduous forest region in the south, the Great Lakes-St. Lawrence region on the eastern border of the Great Lakes, and the boreal forest region in the north (Rowe 1972).

Longer and, most likely, drier climate conditions will affect Ontario's forests directly (Colombo and Buse 1998). However, the indirect impacts of changes in disturbance patterns on forests are possibly even greater. Under future scenarios, variable weather and extreme weather conditions will be more common and fires more frequent in Canada, particularly in the boreal forest, which covers most of Ontario's land base (Stocks et al. 1996, 1998).

In terms of biotic disturbances, considerable thought has been given to anticipating the impact of climate change on insect populations and associated damage in forestry (e.g., Harrington et al. 2001, Volney and Fleming 2000). In contrast, there has been little discussion of the potential impact of climate change on tree diseases and their subsequent impact on forests, although the link between climate and forest diseases is well known (Hepting 1963). In Ontario's forests, over 20 million cubic metres of wood are depleted annually by diseases, including decay fungi (Gross 1991). Many of these diseases respond to stresses, which make trees more susceptible to infection. In forests, diseases also interact with insects and abiotic stresses to result in forest declines (Manion and Lachance 1992).

Types of Plant Disease

Plant diseases are considered an important component of plant and environmental health and can be caused by infectious or *biotic* pathogens and noninfectious or *abiotic* factors. *Biotic* plant diseases are caused by organisms such as fungi, bacteria, viruses, nematodes, phytoplasmas, and parasitic plants. *Abiotic* diseases are associated with chemical and physical factors, such as temperature or moisture extremes, nutrient deficiencies, mineral toxicities, and pollution.

Biotic Diseases

- *Fungi* cause most biotic plant diseases. Members of this group of non-motile, filamentous organisms lack chlorophyll and absorb nutrients from dead or living organisms. Over 100,000 species of fungi are known, and over 10,000 can cause diseases in plants.
- *Bacteria* are single-celled organisms, most of which decompose organic matter. However, about 100 of the known 1,600 species of bacteria can cause disease in plants.
- *Viruses* are nucleoproteins that are parasitic in plant cells and cause host cells to produce more virus particles. These viruses interfere with host metabolism, causing disease in the host. About 2,000 different viruses have been identified, and about 500 of these cause disease in plants.
- *Nematodes* are microscopic worm-like animals. Several thousand species of nematodes have been identified, and several hundred of these cause plant diseases.

Root-feeding species often decrease the ability of plants to take up water and nutrients, and others produce biochemical reactions when injecting their saliva into the host plant. Removal of nutrients by nematodes typically becomes important only when the nematode population is high. However, wounds caused by feeding nematodes can also act as entry points for other pathogens.

- *Phytoplasmas* are microorganisms without cell walls that live in infected plants and insect vectors. They cause over 200 plant diseases.

Plant Disease Epidemiology

When considering the potential influence of climate change on plant diseases, it is important to understand some of the epidemiological factors that influence how biotic plant diseases initiate, develop, and spread. In general, *primary inoculum* is the initial inoculum that starts an epidemic in each crop or year. The different types of primary inoculum are often classified by method of survival or overwintering (soilborne, debris- or hostborne, vectorborne, introduced). Some pathogens have more than 1 type of primary inoculum. Soilborne plant pathogens survive in soil apart from their host or host debris, and soil is the source of primary inoculum. Debris- or hostborne pathogens overwinter in infected tissues of perennial plants and/or in plant debris of annual plants. Primary inoculum of these pathogens is produced from these locations in the next growing season. Vectorborne pathogens are carried and transmitted to host plants by organisms such as insects and nematodes, which are often an integral component of the pathogen's life cycle. Introduced pathogens typically do not overwinter in Ontario's climate and are introduced in seed or on air currents from the south.

Monocyclic diseases produce only primary inoculum and thus typically have only one infection cycle each year. However, many of the more economically important pathogens produce secondary inoculum during the growing season, resulting in multiple infection cycles each season. With these *polycyclic* diseases, the number of cycles each year is restricted by the pathogen, duration of the season, and prevailing environmental conditions. Secondary inoculum is produced by many pathogens from existing diseased tissues, and this inoculum continues to spread the epidemic within a particular crop. The amount of secondary inoculum produced and subsequent infection can be summarized by calculating the rate of disease spread within each crop during a growing season.

A diagram of the disease cycle for biotic plant diseases is provided in Figure 1. After the primary inoculum infects the host, the plant begins to exhibit disease symptoms. The pathogen, in the case of polycyclic diseases, produces secondary inoculum, and then (or near the end of the growing season) produces survival structures. These structures allow the pathogen to survive winter dormancy, and they produce primary inoculum the next season.

Biotic Plant Diseases and Environment

Environmental factors dramatically affect the development of plant diseases. Plant pathologists often use a disease triangle (Figure 2) to illustrate the intimate relationship among plants, pathogens and the environment. For a plant disease to develop, a susceptible host, a virulent pathogen, and a suitable environment must occur simultaneously. Because of this intimate relationship among plants, pathogens,

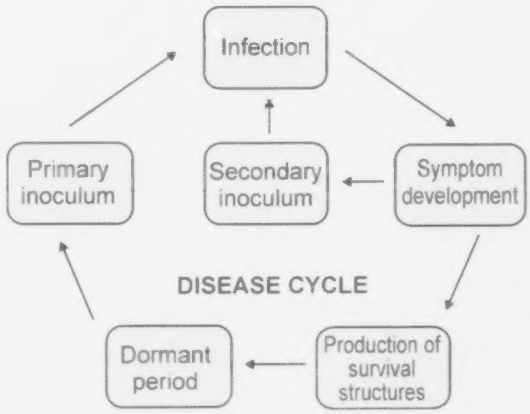

Figure 1. *A typical disease cycle of a plant pathogen, illustrating the relationships between survival and production of primary and secondary inoculum (adapted from Agrios 1997).*

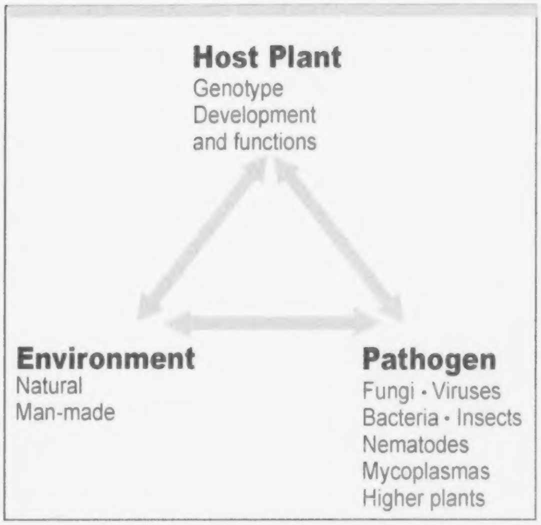

Figure 2. *The disease triangle, illustrating the relationships among host, pathogen and environment in the development of plant disease.*

the environment, climate change is expected to affect the incidence and severity of plant diseases in Ontario and elsewhere.

Typically, the 2 most important environmental factors in the development of plant disease epidemics are temperature and moisture. In temperate regions such as Ontario, most plant pathogens are not active in late fall, winter, and early spring because of low temperatures. Some diseases are favoured by cool temperatures, while others are favoured by moderate or hot conditions. Disease often occurs when temperatures are more stressful for the plant than for the pathogen. Moisture, in the form of free water or high humidity, is necessary for many pathogens to infect, reproduce, and spread, although some can cause disease in dry conditions. Plant diseases require varying environmental conditions to develop; thus, it is vital to understand the environmental requirements of individual plant pathogens before predicting responses to climate change.

Abiotic Diseases

Abiotic plant diseases are caused by noninfectious factors such as nutrient deficiencies, air pollutants, and temperature and moisture extremes. Abiotic diseases can affect plant health directly and indirectly. Direct effects include the development of symptoms related to a deficiency or excess of a physical factor such as moisture, heat, or nutrients, as well as toxic effects caused by inappropriate use of pesticides or chemical pollutants to which plants are intentionally or unintentionally exposed. Abiotic diseases can indirectly affect plant health by weakening their defense mechanisms, thereby predisposing plants to infection by pathogens.

Interactions Between Biotic and Abiotic Diseases

Several important plant diseases are initiated by abiotic stress. Forest decline diseases, for example, are caused by a combination of plant predisposition and a repetitive sequence of plant stresses that weaken a plant until it becomes susceptible to weak pathogens that often infect and kill the plant. In temperate climates, plants that are stressed by biotic or abiotic factors during a growing season are often predisposed to freezing damage during the subsequent winter.

Climate Change and Plant Disease: An Overview

The scientific literature provides some background on the potential impact of climate change on plant diseases. Much of the literature focuses on diseases of agricultural crops and includes discussion of the influence of temperature, precipitation, CO_2, ozone, ultraviolet light, and insects on plant disease.

Temperature and Precipitation

The large population size and short generation time of plant pathogens are expected to make them the first organisms to show effects of climate change (Chakraborty et al. 1998b). In northern latitudes, the impacts of plant pathogens are expected to increase with warming, because low temperatures and long winters currently reduce the survival, generations per year, reproduction rate, and activity of most pathogens attacking crops during the growing season (Harvell et al. 2002, Kaukoranta 1996). However, climate change may bring positive and neutral as well as negative impacts on plant diseases (Coakley 1995, Chakraborty et al. 1998a). Changes will occur in the type, amount, and relative economic importance of pathogens and diseases, altering the disease spectrum, particularly for pathogens with alternate and alternative hosts. Impacts will vary depending on host, pathogen, and changes in climate (Chakraborty et al. 1998a).

Harvell et al. (2002) considered the consequences of warmer temperatures on host-pathogen interactions and concluded that there will be 3 main effects: 1) increases in pathogen development rate, transmission, and generations per year, 2) increases in overwintering of pathogens, and 3) changes in host susceptibility to infection. Furthermore, they suggested that the most severe and unpredictable consequences would occur if populations of pathogen and host, which were formerly geographically separated due to climate constraints, converged.

With changes in climate, plants will migrate to new areas, and their pathogens will follow. How quickly pathogens migrate to follow host plants will depend on factors such as their dispersal mechanisms, suitability of the environment for dispersal to occur, survival between seasons, and changes in host physiology and ecology in the new environment. If a host is chronically stressed due to less than optimum conditions, its health would deteriorate and its susceptibility to disease would increase, particularly in perennials (Chakraborty et al. 1998a). New diseases may establish in a region, while some established diseases may cease to be economically important. Although climate changes may reduce the suitability of a crop for a region, it may continue to be grown for agro-ecological or economic reasons.

Climate change can modify host physiology and resistance and alter stages and rates of pathogen development (Coakley et al. 1999). It can also affect disease management by altering efficacy of biological and chemical control options (Chakraborty et al. 1998a, Coakley et al. 1999). For example, heavy rains reduce fungicide residue. Crop plants growing under elevated CO_2 could be altered morphologically or physiologically, affecting uptake, translocation, and metabolism of systemic fungicides. For example, increased thickness of the epicuticular wax layer on leaves could result in slower and/or reduced uptake by the host, while increased canopy size could negatively affect spray coverage. Conversely, if higher temperatures increase plants' metabolic rates, they may take up chemicals more quickly, which may result in greater toxicity (Coakley et al. 1999).

The nature and magnitude of global climate change could influence plant diseases and the efficacy of management options, in turn affecting the productivity and sustainability of agricultural systems. While a shift in the mean temperature or rainfall may appear to affect plants marginally, the effects are greatly magnified at extreme values. Thus, the impact of such events is particularly important to understand. With this shift, the hydrological cycle will likely intensify and climate variability will likely increase. An increase in rainfall intensity could greatly affect agriculture and plant pathogens in particular. Such effects will interact with changes in plant morphology and chemistry brought about by enhanced CO_2 concentrations (Chakraborty et al. 1998a).

Carbon Dioxide

CO_2 concentrations in the troposphere are projected to increase from 355 ppm (v/v) to 710 ppm by 2050. Higher CO_2 concentrations will likely result in increased biomass production due to increased water use efficiency. Little is known about the effect of higher CO_2 concentration on incidence and severity of plant diseases. Increases in CO_2 from 0.03 to 0.07% may have a slightly stimulatory effect on growth of pathogens (Manning and Tiedemann 1995).

The impact of increased CO_2 concentrations on plant diseases will likely be through changes in host physiology and anatomy. Some significant changes that will likely influence plant disease severity under elevated CO_2 are lowered nutrient concentration, leading to partitioning of nitrogen from photosynthetic proteins to metabolism that is limiting to plant growth; greater carbohydrate accumulation in leaves; more waxes, layers of epidermal cells, and fiber content; production of papillae and accumulation of silicon at the sites of appressorial penetration; and increased mesophyll cells (Chakraborty et al. 1998a).

Elevated CO_2 is expected to increase canopy size and density (Coakley et al. 1999), resulting in greater biomass of high nutritional quality, combined with higher microclimate humidity (Manning and Tiedemann 1995). These changes will likely promote foliar diseases such as rusts, powdery mildews, leaf spots, and blights (Coakley et al. 1999, Manning and Tiedemann 1995); however, Karnovsky et al. (2002) did not observe an increase in infection by poplar leaf rust under elevated CO_2 except in the presence of high ozone. Under elevated CO_2, increased partitioning of assimilates to roots occurs consistently in crops such as carrot (*Daucus carota*), sugar beet (*Beta vulgaris*), and radish (*Rhaphanus sativus*). If more carbon is stored in roots, losses from soilborne diseases of root crops may be reduced under climate change (Coakley et al. 1999). The inoculum potential of non-biotrophs, from more abundant crop debris, would increase (Manning and Tiedemann 1995). Evidence indicates that high-CO_2 leaf litter decomposes at a slower rate (Coakley et. al 1999). Increased plant biomass, slower litter decomposition, and higher winter temperatures could increase pathogen survival on overwintering crop residues and increase the amount of initial inoculum available to infect subsequent crops (Coakley et al. 1999).

Normal soils may contain as much as 6-18% CO_2, depending on organic matter decomposition, microbial and root respiration, and other factors. Most soil-inhabiting fungi tolerate more than 10 or 20 fold increases in atmospheric CO_2 concentration. Some typical soilborne plant pathogens, such as species of *Phytophthora*, *Aphanomyces*, and *Sclerotium*, and pathotypes of *Fusarium oxysporum* are well adapted to and even multiply better at high CO_2 and low oxygen levels (Manning and Tiedemann 1995).

In recent studies on host-pathogen interactions in selected fungal pathosystems, 2 important trends have emerged related to the effects of elevated CO_2. First, initial pathogen establishment may be delayed because of changes in pathogen aggressiveness and/or host susceptibility. Second, pathogens were more fecund under elevated CO_2 (Coakley et al. 1999). The combination of increased fecundity and conducive microclimate within enlarged canopies may provide more opportunities for severe infection. For genetically diverse pathogens, an increase in population size and number of generations in conducive microclimates may lead to the development and proliferation of well adapted and possibly more destructive sub-populations (Chakraborty et al. 1998a).

Ultraviolet Light

Ultraviolet (UV) light has long been known to influence plant pathogenic fungi. This light may stimulate spore production in a wide range of fungi, but may also reduce spore survival during dispersal or early stages of infection (Paul et al. 1998). Although an increase in solar UV-B radiation due to ozone depletion could promote sporulation of pathogenic fungi in a way that could greatly increase the frequency and intensity of epidemics, normal daylight already contains enough UV light to stimulate sporulation of light-dependent fungi. Provided that this stimulation is mainly a qualitative effect of specific wavelengths, a slight quantitative increase in UV-B radiation is not likely to influence the life cycle of pathogenic fungi greatly (Manning and Tiedemann 1995).

Ozone

Ozone is considered to be the most phytotoxic of the common air pollutants. It can cause chlorotic and necrotic lesions on sensitive plant species, and even in the absence of visible symptoms, photosynthesis and growth can be inhibited. Ozone damage can lead to reduced competitive fitness of plants, and reduced vitality makes plants more susceptible to plant pathogens (Sandermann 2000). Direct effects of ozone on fungal pathogens are not significant (Manning and Tiedemann 1995), although interactions between ozone damage and infection by *Alternaria solani*, the causal agent of early blight of potato, have been reported (Holley et al. 1985a, 1985b). Ozone-induced metabolic changes can apparently persist in plants over days or months. Researchers have reported both increased (Sandermann 2000) and decreased (Coleman et al. 1988) disease susceptibility in plants after ozone exposure. According to Tiedemann and Firsching (1998), ozone effects on plant disease susceptibility may be strongly altered by interfering factors such as plant developmental stage, nutrient supply, and other atmospheric trace gases.

Insects

Insects are important vectors of many plant diseases. The potential effects of climate change on agricultural insect pests have been explored by Porter et al. (1991). Temperature is the most critical climatic variable, with low temperatures often being more important than high temperatures in determining global insect species' distributions. Winter is the most critical time for many insect pests because low-temperature extremes can significantly increase mortality, thereby reducing population levels in the following season. Information from general circulation models indicates that future temperature changes will be greatest in winter. Predicted higher winter temperatures could increase the survival of many overwintering species and allow them to overwinter even farther north. For example, in northern temperate zones, aphid (Aphididae) damage is expected to increase due to earlier aphid activity in the spring, increased development rate, and greater survival over winter. Serious outbreaks of aphid-vectored viruses have been observed following mild winters (Harrington et al. 1995).

For all insect species, higher temperatures that are below the species' upper lethal limit could reduce the time it takes to reach reproductive maturity and, therefore, increase pest populations more quickly. Such effects would be particularly important at higher latitudes, where current temperatures, particularly in the spring, limit insect development and distribution.

Increases in food availability as a result of plant stress could dramatically affect how quickly pest populations develop. Pest outbreaks are more likely to occur if plants are stressed (e.g., drought stress) because their defensive systems are compromised and resistance to pest infestation lower. Drought may become more frequent and severe in the future if increased evapotranspiration rates associated with predicted higher temperatures are not compensated sufficiently by increases in precipitation.

Climate Change and Plant Disease: An Ontario Perspective

In assessing the potential impact of climate change on plant diseases in Ontario, we considered the impact of climatic assumptions on 3 key stages of the disease cycles of the most prevalent and economically important diseases of agricultural and forestry species in Ontario. The disease stages that were assessed were 1) survival of primary inoculum of the pathogen, 2) rate of disease progress during a growing season, and 3) the duration of the annual epidemic in relation to the host plant. The effect of climate change on each of these components, and the anticipated effect this would have on disease severity, were assessed for each disease. We included minor diseases when evidence suggested that their relative prevalence or severity could change due to predicted climate change. These results are summarized in Appendix 1. This assessment does not take into account alien pathogens although the potential for new introductions under a changing climate is well known (Baker et al. 2000). In many cases, our assessments were necessarily subjective. To illustrate these assessments, several case studies were selected and are presented in more detail in the following sections.

Under climate change, the duration or length of epidemics is expected to increase or remain the same, depending on the host crop plant. Some crops, such as corn (*Zea mays*) and soybeans (*Glycine max*), will be modified to take advantage of the longer growing season, resulting in greater yields, but also longer disease epidemics. For other crops, such as fresh market vegetables, more growing time may not be an advantage, because early maturation is often a more valuable market advantage than increased yield. However, overlapping planting dates within the same field may still effectively prolong the presence of susceptible hosts and, therefore, prolong the duration of disease epidemics.

Fungal Diseases

Plant pathogenic fungi have several primary inoculum types:

- *Soilborne* fungi survive in soil by producing structures such as sclerotia or thick-walled spores (e.g., chlamydospores or oospores). Soilborne fungi include species of *Botrytis, Fusarium, Phytophthora, Pythium, Rhizoctonia, Sclerotinia* (Case Study 1), *Sclerotium*, and *Verticillium*. In our present climate, survival structures produced by soilborne fungi can survive for years in soil. Therefore, the milder winters and reduced soil moisture expected in Ontario with climate change should not greatly affect the survival of these fungi.
- *Host-* or *debrisborne* fungi overwinter and produce primary inoculum on infected tissues of perennial plants and on plant debris of annual plants. Fungi of this type include *Alternaria, Cercospora, Colletotrichum, Erysiphe, Phaeoisariopsis* (Case Study 2), *Phomopsis, Septoria*, and *Venturia*. Milder winters in Ontario should increased survival of these pathogens.
- Several important introduced fungal pathogens do not survive Ontario winters and are *seedborne*, or *airborne* from the south. These fungi include *Peronospora tabacina, Puccinia* spp. on cereals, and *Ustilago tritici*. Milder winters in Ontario may allow some of these pathogens to overwinter or to migrate earlier, resulting in earlier infection and longer epidemics.

Case Study 1 - *Sclerotinia sclerotiorum*

A soilborne fungal pathogen with a wide host range, including many crops in Ontario.

Sclerotinia sclerotiorum is a fungal plant pathogen that can cause disease in more than 400 plant species. In Ontario, S. sclerotiorum is particularly severe in bean *(Phaseolus vulgaris)*, canola *(Brassica* spp.), carrot, lettuce *(Latuca sativa)*, and soybean but, depending on environmental conditions, can also be severe in potato *(Solanum tuberosum)*, tomato *(Lycopersicon esculentum)*, and forage crops such as alfalfa *(Medicago sativa)* and clover *(Trifolium* spp.). Crop losses ranging from 25 to 50% are commonly reported in various crops in severe, localized outbreaks; however, on average, crop losses of 2-7% are a reasonable estimate in bean, canola, and soybean. Given the economic value of these crops in Ontario in 2000, such crop losses would be valued at annual losses of up to $40 M (farm value at 7% loss).

S. sclerotiorum is a soilborne fungus that spends at least 95% of its life cycle in and on soil as sclerotia, which are small, black survival structures that resemble tiny pebbles. Under suitable conditions, sclerotia germinate to produce fruiting structures called *apothecia*, which release ascospores into the air. These ascospores initiate disease; to do so, they require senescent plant tissues, such as flower petals or older leaves.

Environmental variables, such as temperature and soil moisture, play a critical role in the ability of *S. sclerotiorum* to survive in soil. Factors such as repetitive freezing and thawing contribute to the death of sclerotia, particularly when directly exposed on the soil surface without prolonged snow cover. Biological factors, such as parasitic fungi and fungal-feeding insects and nematodes, are also an important factor in the survival of sclerotia. The activity of these organisms is, in turn, also affected by temperature and soil moisture.

For *S. sclerotiorum* to cause disease, conditions must be conducive for apothecia production and then ascospore infection. These stages must coincide with a growth stage of the plant during which senescent tissues are sufficiently present. Prolonged periods of moist soil and cool soil temperatures that occur once the crop canopy has at least partially closed favour apothecia production. Infection by ascospores occurs at cool to moderate temperatures and requires moisture. These conditions must be prolonged for disease to spread.

Sclerotia are very hardy survival structures. Milder winters may increase survival, while increased freeze/thaw cycles and microbial activity may decrease survival. Therefore, with changes in climate, sclerotia survival over winter may not change significantly. Warmer, drier growing seasons will result in an environment that is less conducive for apothecia production and infection by ascospores. Overall, if Ontario's climate changes as expected, severity of diseases caused by *S. sclerotiorum* should decrease.

Case Study 2 - Angular Leaf Spot of Bean

A debrisborne fungal disease that was recently introduced to Ontario.

Angular leaf spot of bean (ALS) is caused by the fungal pathogen *Phaeoisariopsis griseola* (Sacc.) Ferr. ALS is primarily a disease of the tropics and subtropics and is considered to be of minor importance in most bean-producing areas of the northern United States, although it can cause epiphytotics when conditions are suitable. In the northern states, yield losses of 50% have been reported. ALS was first reported in Canada in 2000 on snap beans, and it overwintered on crop debris, causing disease in 2001.

ALS overwinters as *stromata*, which are compact masses of fungal mycelium, in host debris. Stromata produce microscopic, tree-like structures called synnemata, which in turn produce the primary inoculum. Synnemata formation occurs in humid conditions (>85% RH) and is completed in about 24 hours at 24°C. Spores are released from synnemata in dry atmospheric conditions and are spread by air currents, rain splash, and insects. Spores infect bean plants causing spots on leaves, stems and pods. High levels of infection on leaves can cause defoliation. Premature defoliation and pod lesions reduce yields. The fungus forms stromata on lesions. Stromata are hardy and can stay dormant until conditions are suitable for secondary spore production. ALS is a polycyclic disease, with secondary inoculum of the pathogen contributing to at least several disease cycles in a season. The most serious epidemics of ALS occur when moderate temperatures and periods of rain or high relative humidity, long enough to induce synnemata formation and infection, alternate with dry, windy conditions.

Primary inoculum for ALS is debris- and seedborne; however, overwintering fungal tissue in host debris is considered the main source of primary inoculum. Milder winters should result in increased survival of ALS. In addition, ALS survives better in surface debris than when buried. Higher temperatures and greater evaporation are expected to result in greater use of conservation tillage and no-till to conserve soil moisture, which means more debris on the soil surface. Therefore, although ALS is a tropical/subtropical disease, its ability to overwinter in Ontario is expected to increase with climate change.

Rate of disease progress for ALS in a changing climate is difficult to assess because it has only just established in Ontario. This pathogen's requirement of rain or high humidity for spore production and for infection suggests epidemics that start in the moist spring and early summer will slow during the hotter, drier summer months and resume again in the late summer.

Growing season length is an important factor in epidemics of polycyclic diseases. Bean growers are expected to take advantage of longer growing seasons, resulting in longer epidemics, higher final disease incidence and yield loss, and increased stromata to overwinter and produce primary inoculum in the next season.

If Ontario's climate changes as expected, ALS could become a significant bean disease due to increased survival over milder winters and a longer season for epidemics to develop.

Monocyclic fungal diseases, such as those caused by *Ustilago* and *Sclerotinia* species, should not be as affected by climate change as polycyclic diseases. With polycyclic diseases, such as those caused by *Colletotrichum*, *Peronospora*, *Phytophthora* and *Puccinia* species, each disease cycle multiplies inoculum many fold, so a longer growing season in Ontario should result in more disease cycles and inoculum. However, monocyclic vascular wilts and root rots will result in more severe disease symptoms as the result of increased drought stress.

Moisture, in the form of rain, dew or high humidity, is the most important factor in the development of most epidemics caused by fungi. High levels of moisture promote sporulation and facilitate spore release and germination, and prolonged or repeated high moisture conditions lead to epidemics. Most fungi are active over a range of temperatures, but activity is highest at moderate temperatures (18-24°C). With adequate moisture and optimum temperatures, polycyclic fungi can complete their disease cycles quickly, usually within a few days. Overall, if summers are warmer and drier, plant diseases caused by fungi should decrease. Disease levels may change little in the spring and fall when moisture is more abundant, but disease progress should slow in the warmer, drier summer months. If disease cycles for polycyclic diseases are reduced, and the progress of monocyclic diseases slows, primary inoculum for the next season will decrease. Some

Case Study 3 - Stewart's Wilt of Corn

A bacterial disease, spread by an insect vector, that is becoming more prevalent in Ontario.

Stewart's wilt of corn is caused by the bacterium *Pantoea stewartii*. The predominant vector in Ontario is the corn flea beetle (*Chaetocnema pulicaria*). The bacterium overwinters in the digestive tract of beetles. The cold winters in Ontario usually kill most beetles; however, occasional outbreaks of this disease have occurred in Ontario since 1932, and the mild winters in 1998 and 1999 led to the most severe outbreak of Stewart's wilt recorded in Ontario.

Primary inoculum depends on survival of flea beetles, and the number of flea beetles emerging from hibernation in spring depends on winter temperatures. The numbers of emerging adults can be estimated by averaging the mean temperatures for December, January, and February (Eastburn 1996). Zero, 1, 2, and 3 months above - 4.4°C suggest minimal, low, moderate to high, and high risk of Stewart's wilt, respectively (Esker and Nutter 2002). Snow or other winter cover likely has little effect in enhancing survival of overwintering flea beetles (Eastburn 1996).

Primary inoculum is spread when infested beetles that survive the winter feed on corn seedlings in spring. Non-infested flea beetles feed on infected plants and then become vectors. During the growing season, new generations of flea beetles become infested, greatly increasing the number of infested insects. Once infested, flea beetles carry and transmit the pathogen for their entire lifetime. Beetles can migrate or be carried by wind 20 miles or more. Prolonged periods of wet summer weather are unfavourable for beetle multiplication and feeding, while dry weather is favourable (Eastburn 1996).

Plants affected early in the season (before plants reach 60 cm tall/5 leaf stage) are most severely affected and suffer a 40-100% yield loss. Plants infected later in the season usually have less than 10% yield loss. Sweet corn and inbred lines used for seed production are most susceptible to Stewart's wilt. High temperatures generally increase disease severity (Hershman et al. 2002).

Growing resistant hybrids is considered the best way to control Stewart's wilt. After mild winters, early applications of insecticides to kill corn flea beetles can also be helpful. Excessively high levels of nitrogen and phosphorous should be avoided.

If winter temperatures increase in Ontario, more corn flea beetles will overwinter. Anticipated drier summers would also favour these beetles, which do poorly in prolonged wet periods. In addition, the expected higher temperatures will likely increase severity of Stewart's wilt. Thus, if Ontario's climate changes as predicted, the occurrence and severity of Stewart's wilt likely will increase.

fungal plant pathogens, such as *Podospherea*, *Sphaerotheca*, *Uncinula*, and *Ustilago* species, thrive in warmer, drier conditions, and these should increase in significance in Ontario if the climate changes as expected.

Bacterial Diseases

Soilborne bacterial plant pathogens, such as *Agrobacterium tumefaciens*, build up their populations in host plants and are released into the soil, where they can survive to act as primary inoculum the next season. Host- and debrisborne bacteria survive in host tissues. On perennial hosts, bacteria, such as *Erwinia amylovora* on apple (*Malus* spp.), overwinter on infected host tissue, and primary inoculum is spread from host to host the next season. On annual hosts, bacteria, such as *Pseudomonas syringe* pv. *phaseolicola*, survive in host debris in soil or on the soil surface. Vectorborne bacterial pathogens, such as *Pantoea stewartii* (Case Study 3), survive in insect vectors, and these vectors act as the source of primary inoculum the next season. Introduced bacterial pathogens, such as *Xanthomonas campestris*, *Pseudomonas syringae* pv. *tomato*, and *Xanthomonas axonopodis* pv. *vesicatoria* (Case Study 4), come from infected seed and perhaps survive in debris, soil, and weeds. Milder, shorter winters will likely have little effect on soilborne bacterial pathogens; however, survival of host- or debrisborne and vectorborne primary inoculum is expected to increase. The effect on primary inoculum of introduced pathogens cannot be estimated.

Case Study 4 - Bacterial Spot and Speck of Tomato

Common bacterial diseases of tomato in Ontario.

Bacterial speck caused by *Pseudomonas syringae* pv. *tomato* and bacterial spot caused by *Xanthomonas axonopodis* pv. *vesicatoria* are 2 diseases of tomato that have become increasingly problematic for growers throughout North America. In Ontario, both pathogens are commonly introduced on contaminated seed (Louws et al. 2001) and may also survive in soil, on plant debris, or on other weedy hosts, as reported for the southern U.S. states (Jones et al. 1986, McCarter et al. 1983). Healthy tomato transplants have varying levels of these bacteria growing epiphytically on their surface, but disease severity in the field depends more on environmental conditions than on the population size of epiphytic bacteria at the time of transplanting (Cuppels and Elmhirst 1999). In the field, the size of the epiphytic population can increase rapidly if conditions are favourable. Both pathogens require high moisture and relative humidity for multiplication, but warm temperatures are optimal for spot development, whereas cool temperatures favour tomato speck. Typically, these epiphytes spread from the original infested transplants via wind and rain (Bernal and Berger 1996) and then will establish infections if introduced into the leaf interior by wounding or by driving rain. Once lesions formed by either pathogen establish on foliage, they can continue to produce inoculum for extended periods.

In southwestern Ontario, these 2 diseases occur on both processing and fresh market tomatoes (1998 value = ~$78 million). Although seed treatment, sanitation, and crop rotation are used to decrease overwintering inoculum, the primary management strategy continues to be the use of copper-based bactericides. Despite frequent reports of copper-resistant strains of the bacteria, the bactericides are still reasonably effective. Efforts are underway to incorporate chemically induced plant disease resistance into the management strategies by applying Actigard 50WGâ or CGA245704 plant activators at 10-day intervals (Louws et al. 2001). Despite these management efforts, losses caused by tomato spot and speck vary greatly from year to year. Tomato spot alone is estimated to have reduced the 1990 and 1991 Ontario crops by an average of 2 tons/acre (~$215/acre), as well as causing a loss in quality (Dick 2002, pers. comm.[1]).

If Ontario's summers become warmer and drier, the severity of bacterial spot and speck could decrease because they require moisture and high humidity. However, this reduction may be counteracted by enhanced survival of the bacteria in crop residue and soil and as epiphytes on weeds during the milder winters. The role of severe rain and wind storms in facilitating the entrance of these bacteria into the leaf, and the subsequent initiation of disease, will probably be more critical to crop losses due to speck and spot. If the relative humidity early in the season, or later within the canopy, is high enough to allow sufficient epiphytic growth of these pathogens, the increased frequency and severity of such storms will increase the likelihood of severe disease episodes. Overall, the incidence of disease may be lower, but when infection does occur, damage could be great. Currently, there is no "after-infection" method of control; thus, growers may be compelled to continue routinely using bactericides and/or plant defense activators in anticipation of a severe storm. The alternative is to gamble that the appropriate combination of epiphytic growth and severe storms will not occur. More research is needed to develop a forecasting system, based on monitoring conditions for epiphytic growth of the pathogens, to allow growers to safely reduce the number of bactericide applications.

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Bacterial diseases are considered polycyclic. Bacteria are spread to their host plants mainly by water, usually in the form of rainsplash, and by insects. Moisture is the most important factor in the development of bacterial diseases. Abundant moisture increases multiplication, oozing, and spread of bacteria. In humid, wet conditions, infected plant tissues can exude masses of bacteria that are spread from host to host by rainsplash and insects. Therefore, the warmer drier summers expected under climate change in Ontario should limit bacterial diseases. However, bacteria often enter their plant hosts through wounds, and the expected increase in frequency and intensity of summer storms with high winds, rain, and hail will increase plant wounding and provide moisture for spread of bacteria.

Viral Diseases

Primary inoculum for plant diseases caused by viruses can be hostborne and overwinter in perennial hosts (e.g., plum pox virus). Introduced viruses arrive in seed (e.g., bean common mosaic virus, soybean

Case Study 5 - Potato Leaf Roll Virus

An insect-vectored viral pathogen of potato and other crops in Ontario.

The severity of diseases caused by plant viruses is determined primarily by the amount of inoculum and time of infection. The amount of virus inoculum is influenced by winter survival of its (alternate) plant host, winter survival of its transmission vector, the vector's ability to transmit the virus throughout its life (persistent), and the rate of accumulation and spread of the transmission vector. Infection of plants at an early developmental stage usually leads to more severe disease symptoms. For some viruses, higher temperatures can also result in more severe disease.

Potato leaf roll virus (PLRV) infects many plants, including potato, tomato, tobacco (*Nicotiana tabacum*), bean, alfalfa, onion (*Allium* spp.), apple, strawberries (*Fragaria* spp.), turf, and peach (*Prunis persica*). PLRV belongs to the luteovirus genus of plant viruses and is transmitted by a variety of aphids, in particular the green peach aphid (*Myzus persicae*). Newly hatched aphids are virus-free and must acquire the virus by feeding on a PLRV-infected plant, such as volunteer potatoes. After an aphid has acquired the virus, it can transmit the virus for the rest of its life in a circulative or persistent manner. The virus can be spread among plants within a field, and between fields, by the winged forms of the aphid, but much of the spread within a field is accomplished by the wingless forms (Brunt et al. 1996). Once the virus has been introduced to a plant by feeding aphids, it spreads from leaves to tubers. Crops planted with these tubers can be severely stunted, resulting in devastating yield losses and poor tuber quality.

A large percentage of potato leaf roll disease (PLR) was found to be related to both the amount of primary inoculum and the thermal unit accumulation within a crop (Thomas et al. 1997, DiFonzo et al. 1994). The latter is due to the effect of temperature on development and flight of winged forms of the aphids.

The milder winters that should result from climate change in Ontario are likely to increase primary inoculum by increasing winter survival of infected host plants. In addition, more aphid vectors will survive the winter, and they will aquire the virus and infect plants earlier in the year. Also, under warmer temperatures, more aphids will develop over a longer period, and winged forms will find optimal temperatures for flight more often. Therefore, if Ontario's climate changes as predicted, PLR will likely increase.

mosaic virus) or in tissues of plants used for vegetative propagation (e.g., potato leafroll virus, plum pox virus).

Although vectors do not play a role in primary inoculum of viruses, they are very important in the spread of viral diseases. Virus vectors include insects, especially aphids (e.g., soybean mosaic virus, bean common mosaic virus, plum pox virus, bean yellow mosaic virus, potato leaf roll virus (Case Study 5), and cucumber mosaic virus), and nematodes (e.g., tobacco ringspot virus). Mechanical injury is also an important way that viruses can spread between plants (e.g., tobacco mosaic virus).

The severity of viral diseases is determined largely by the amount of inoculum and time of infection. The amount of virus inoculum is influenced by winter survival of its (alternate) plant host, winter survival of its transmission vector, the ability of the virus vector to transmit the virus throughout its life (persistent), and the rate of accumulation and spread of the transmission vector. Plants infected at an early developmental stage usually have more severe disease symptoms. For some viruses, higher temperatures cause stronger symptom development.

If climate change results in milder winter temperatures in Ontario, insect vectors such as aphids are expected to have increased survival, while higher summer temperatures will increase their development and reproductive rates. Milder winters are also expected to increase survival of alternate weed hosts of viruses. Increases in frequency and intensity of summer storms with high winds, rain, and hail will increase plant wounding and result in increased transmission of viruses via mechanical means. In addition, for some viruses, warmer temperatures result in increased symptoms. Therefore, if Ontario's climate changes as predicted, viral diseases of plants should increase in importance.

Case Study 6 - Soybean Cyst Nematode

The soybean cyst nematode (*Heterodera glycines*, SCN), first detected in Ontario in 1987, is the major economically limiting disease problem of soybeans in North America. SCN is a typical cyst nematode with eggs surviving over winter in a durable melanized cyst that is formed from the body of the female nematode. The nematode larvae hatch from the eggs within the cyst and emerge as second-stage juveniles that are attracted to actively growing soybean roots and penetrate the plant near the root tip. Once in the root, the larvae start feeding in the vascular system and remain there through 3 molts to become adults. The adult male, slender, motile and worm-like, stops feeding and exits the root. The lemon-shaped adult female protrudes from the root and mates with the free-swimming males. Some of the fertilized eggs are released immediately as a gelatinous mass, and these eggs give rise to new generations of larvae in that growing season. The remaining eggs (several hundred) are retained in the female body, which matures into a cyst that can survive up to 9 years in the soil.

Reduced yield caused by feeding nematodes is largely due to decreased root growth and the consequent reduction in water and nutrient uptake. Reduced nodulation by nitrogen-fixing bacteria also results in reduced nitrogen fixation. Often no symptoms, other than reduced growth, are seen, and this reduction may be overlooked if the entire crop is infected. In more extreme situations, symptoms typical of nutrient and water deficiencies appear, further reducing yields. Disease management involves appropriate rotation with a crop that is not attacked by SCN, e.g., corn and wheat, and the use of resistant cultivars. These cultivars restrict the reproduction of susceptible nematodes but can select for new races of SCN that can develop and reproduce on the resistant cultivars. To prevent new races from developing, resistant and susceptible cultivars must be rotated.

Various stages in the SCN disease cycle are affected differentially by temperature and moisture. The highest winter survival of SCN eggs occurs in the colder areas of the continent (Nematology Committee of the Southern Soybean Disease Workers 2002). Thus, spring inoculum levels may be highest in the northern range of soybean culture. Optimal soil temperatures for egg hatch, root penetration, and juvenile and adult development are 24°C, 28°C and 28–32°C, respectively; below 15°C and above 35°C little development occurs (Chen et al. 2001). Thus, temperature can affect the number of SCN generations per growing season. In theory, with fewer generations, new races will develop less quickly.

In Ontario, predicted temperature increases could minimally affect yield loss caused by SCN. The more moderate winter temperatures will reduce egg survival, while the higher temperatures in the growing season will increase egg hatch, the rate of nematode development, and perhaps, the number of generations per season. Soil water is important for SCN movement and development, but water is unlikely to be a limiting factor early in the season. More importantly, the drier growing conditions of summer will increase the yield loss due to SCN because of reduced root surface.

Nematodes

Most plant pathogenic nematodes spend part of their lives in the soil, making soil the primary inoculum source. The life cycle of a nematode, egg to egg, can develop within 2 to 4 weeks under optimum environmental conditions. Temperature is the most important factor, and development is slower with cooler soil temperatures. Overwintering of nematodes should not be significantly affected by predicted changes in climate, although for some, such as the soybean cyst nematode (*Heterodera glycines*) (Case Study 6), mild winters may reduce egg viability. Warmer soil temperatures should accelerate nematode development in Ontario, perhaps resulting in more generations per season, and drier temperatures should increase symptoms of water stress in plants infected with nematodes such as the soybean cyst nematode (Case study 6). Overall, if Ontario's climate changes as predicted, diseases caused by nematodes should increase in importance.

Phytoplasmas

Phytoplasmas are plant pathogenic micro-organisms that overwinter in perennial hosts, providing the source of primary inoculum. Insect vectors feed on infected plants, acquire the phytoplasma, and transmit it to healthy plants. In Ontario, aster yellows is the most important disease caused by a phytoplasma (Case Study 7).

Case Study 7 - Aster Yellows

A disease with many hosts, caused by a phytoplasma that is vectored by insects.

Aster yellows is caused by the aster yellows phytoplasma. This pathogen has over 300 hosts in 50 plant families. Cultivated crops that can be infected with aster yellows include carrot, celery (*Apium graveolens*), cucurbits, potato, sage (*Salvia* spp.), tomato, echinacea (*Echinacea* spp.), canola, flax, barley (*Hordeum vulgare*), wheat (*Triticum* spp.), oats (*Avena* spp.), sunflower (*Helianthus annus*) and faba beans (*Vicia faba*). Weed hosts include chicory (*Cichorium intybus*), knotweed (*Polygonum* spp.), lamb's-quarters (*Chenopodium album*), pineappleweed (*Matricaria matricarioides*), plaintain (*Plantago* spp.), quackgrass (*Agropyron repens*), ragweed (*Ambrosia artemisiifolia*), stinkweed (*Thlaspi arvense*), sowthistle (*Sonchus* spp.), wild asters (*Aster* spp.), and wild carrot (*Daucus carota*).

The aster yellows phytoplasma overwinters in perennial hosts, providing the primary inoculum. Leafhoppers (Cicadellidae) are vectors of the pathogen and move the primary inoculum to healthy plants when they feed. Primary inoculum can also be introduced when leafhoppers carrying aster yellows are blown into Ontario from the United States.

If Ontario's winters become milder, more perennial hosts and leafhopper eggs would survive, resulting in more primary inoculum and more vectors for the primary inoculum. Warmer temperatures the rest of the year would result in increased development and reproduction of the vector. In addition, symptoms of aster yellows tend to increase with increasing temperature. Therefore, if Ontario's climate changes as predicted, aster yellows will become a more important disease.

If Ontario's winters become warmer as predicted, more infected perennial hosts and more insect vectors should survive over the winter. In addition, higher summer temperatures will increase vector development and reproductive rates. Therefore, phytoplasmas such as aster yellows should become more important if Ontario's climate changes as predicted.

Abiotic Diseases/Disease Complexes

Plant diseases associated with interactions of biotic and abiotic diseases, or disease complexes, are a unique and important area of consideration for assessing the influence of climate change on plant disease. Forest declines (Case Study 8) are examples of plant diseases that result from a combination of interacting biotic and abiotic factors. They are often referred to by their symptom syndrome, such as *dieback* or *decline*. Such diseases are often characterized by a variety of disease symptoms and signs, are typically scattered in a random pattern throughout a population within a region, and are often host-specific, although more than one tree species in a region may have its own specific decline symptoms. A strong association has already been established between climate change and disease incidence and/or severity in several forest species, including ash (*Fraxinus* spp.), birch (*Betula* spp.), balsam fir (*Abies balsamea*) and maple (*Acer* spp.).

Overall Effect of Climate Change on Plant Disease in Ontario

With significant and relatively rapid changes in climate in Ontario, more new crops and cultivars will be introduced into the province. Adapting agronomic and horticultural cultivars to regional soil and climatic conditions is a well-established practice that involves comparing agronomic performance in multiple locations over multiple years. Only cultivars that perform well, on average, across these locations and environments are selected for commercial use. This process has been highly successful in Ontario for many years and will be instrumental in continuing to select adapted cultivars under expected climate changes. However, this process may not ensure that selected cultivars can withstand the erratic and extreme weather events that are predicted with climate change in Ontario. Plants will need to be bred to tolerate environmental stress, such as drought. Such tolerance will result in healthier crops

Case Study 8 - Maple Decline

A disease complex known to be associated with climate change.

Forest declines are generally viewed as deterioration in tree health over a large area with evidence of decreased growth rate, increasing branch dieback, and above-normal tree mortality. Decline diseases are one of the few examples where a strong association between climate change and disease incidence and/or severity has already been established in several forest species. Episodes of extensive forest declines have been well documented in Europe and North America in ash, birch, balsam fir and maple (Innes 1993, Millers et al. 1989), and a strong relationship was evident between climate warming in the Northern Hemisphere and the onset of crown dieback in 1925, 1937 and 1981 on selected species of northern hardwoods in eastern Canada (Auclair et al. 1992). Increases in forest declines are expected to continue as individual species reach critical dieback threshold temperatures associated with annual increases in temperature.

The most familiar dieback or decline disease in Canada is maple decline, a disease that was first diagnosed in 1913 but received considerable interest during the 1960s because of its putative association with air pollution in eastern North America (Nordin 1954, McIlveen et al. 1986). Recent research has identified that there are actually 3 types of maple declines associated with maples growing along roadsides, in sugarbushes, and in forests. Each maple decline disease is associated with different conditions that occur in these separate ecological locations. Often, it is not a single event or stress that leads to decline diseases such as maple decline but a disease complex of several interacting predisposing (e.g., site characteristics such as soil nutrients or climate), inciting (e.g., several years of excessive drought or precipitation), or contributing (e.g., weak plant pathogens or insect defoliation) factors that progressively stress individual trees until they weaken and die.

Sugar maple (*Acer saccharum*) trees growing in more stressful environments, such as along roadsides and in urban environments, are more likely to be in poor health (Hopkin and Howse 1998). When trees are stressed, they become susceptible to secondary pests such as wood boring beetles, fungal decays and root rots. Climate and weather also play a significant role in causing stress. In a changing climate, increased temperature and evapotranspiration, as well as an increased frequency of extreme weather events such as ice storms or wind storms, will increase stress to forests and potentially increase the frequency of forest declines. Recently, extensive damage to sugar maple forests was reported following an ice storm that affected much of northeastern North America (Smith 2000). In Ontario, over 604,000 ha of hardwood forest, made up largely of sugar maple, were damaged directly by this event (Hopkin et al. 2001b). Ice storm damage or wind events are predicted to become more frequent under climate change models. Such events cause immediate damage but can also predispose trees to fungi and insects, causing additional long-term loss of vigour and mortality (Hopkin et al. 2001a). Ground-level pollutants such as ozone, which have increased in recent years, can also add to these forest stresses (McLaughlin and Percy 1999).

Trees are constantly expanding their range to the limit of their site tolerance. Those on the boundary of their site tolerance are most affected by declines, but declines often occur well within the limit of their natural geographic range. Often individual sites within the range vary as much as those at the edge of the range. Given the predicted changes in climate for Ontario, significant increases in tree decline diseases are anticipated. Increased temperatures and evapotranspiration, as well as increased frequency of extreme weather events such as wind, hail, and ice storms, will increase the frequency and severity of stress factors leading to maple and other forest declines.

that are better able to resist disease and produce improved yields. Also needed are new techniques to develop enhanced resistance.

If Ontario's climate changes as predicted, new plant diseases will appear in the province. The appropriate diagnostic tools, expertise, and personnel must be available to detect new pathogens. The effects of Dutch elm disease, and more recently, soybean cyst nematode and plum pox virus demonstrate how devastating introduced plant diseases can be.

Drier soils and irregular precipitation will result in increased irrigation, which will be economically feasible only with high-value crops such as vegetable, horticultural, and specialty crops. Technology for efficient irrigation could be adapted from those already used in arid climates such as in Israel. Water conservation systems may also Jones, leaf wetness and humidity in the crop canopy and, therefore, contribute to reduced foliar disease compared with more traditional practices such as overhead irrigation. In addition, plants that are not drought-stressed can better defend themselves against infection.

Ontario is predicted to experience more erratic and extreme weather, which could result in an increase in covered crops. In the greenhouse, environmental conditions can be controlled, water can be used more efficiently, and diseases can be better controlled than in the natural environment. However, root diseases that are spread in recirculating hydroponic solutions may become more important.

Climate change and its effects on plant diseases may contribute to some crops becoming nonviable, leading to the replacement of these crops with new alternative crops. Continued research and development will be required to identify alternative crops and to support their implementation in Ontario.

Many native forest pathogens can cause general damage over a wide area as evidenced by root rots (armillaria and tomentosus root diseases), decay fungi, and many of the canker-causing diseases (e.g., hypoxylon canker of poplar [*Populus* spp.]). Ontario's forests are also susceptible to introduced pathogens, such as those that cause Dutch elm disease (*Ophiostoma ulmi*), beech bark disease (*Nectria coccinea* var. *faginata*), and white pine blister rust (*Cronartium ribicola*). These pathogens cause serious damage and threaten the survival of their respective hosts over a broad area. Recently, we have seen the appearance of new pathogens, such as sudden oak death (*Phytphthora ramorum*) in California and Oregon, which could threaten red oak in Ontario given appropriate conditions. All of these diseases, introduced or native, are influenced by climate to varying degrees. Warmer and drier conditions will likely reduce the incidence of diseases such as white pine blister rust and scleroderris canker that require cool, wet conditions for infection and, in the case of scleroderris canker, an extended cold period (Marosy et al. 1989, Van Arsdel et al. 1956). However, warmer and drier conditions will likely increase the damage caused by root rots, particularly annosus root rot (*Heterobasidium annonsum*), the range of which is restricted by a cooler climate. Similarly, diseases such as oak wilt that have seriously affected oak species in the United States (Wilson 2001) and that are found in states adjacent to Ontario would benefit from warmer drier conditions. Facultative pathogens such as *Armillaria* species and wilt diseases such as pine wood nematode and Verticillium wilt, as well as secondary canker-causing fungi, would benefit if heat and drought cause stress in forest and urban trees. The predicted increase in frequency of storms (wind and ice) will make forests more susceptible to breakage and mortality as well as decays. Forest declines could become more extensive and common due to the inciting factors of drought and the potential for variable winter temperatures (e.g., freeze/thaw events).

Due to the long-lived nature of trees and accordingly the limited degree to which they can adapt, potential impacts of climate change must be considered in forest management plans. Issues include species selection, breeding, and site selection. Drought-prone sites might have to be abandoned and short rotation species such as hybrid poplars could become increasingly important for fiber production.

Unlike forest trees, most of the commercially grown crops in Ontario, with the exception of fruit trees, are annuals or short-lived perennials. Thus, appropriate cultivar and cultural practices will evolve as climate change occurs, just as has occurred in Ontario in the past 100 years. However, the rate of change in plant agriculture is expected to accelerate with changes in climate. In the past, continuous input from agronomists, plant breeders, and pest management specialists was essential to the success of plant agriculture in Ontario. This system of research must not be eroded. With the potential effects of climate change, crop research will become more important than ever. We have remained abreast of many changes because of the dedicated work of those who have gone before us. Change will occur more rapidly than ever before, and the research structure must be in place to meet future challenges.

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Appendix 1. Effects of Climate Change on Diseases in Agriculture and Forestry

The predicted effects of climate change on diseases of selected major agricultural and forestry species in Ontario are summarized in Appendix 1. Explanatory remarks were included in the summary table to indicate the most important factors considered for each disease. Reasons for anticipated changes in primary inoculum and disease establishment are listed first, followed by a slash and reasons for anticipated effects on rate of disease progress. The overall estimated effect of changes in climate on each disease is indicated, and a general reference for each disease is provided.

Crop/Disease	Pathogen (Group)[1]	Anticipated Effect of Climate Change (↑,-,↓)[2]			Remarks[4]	Estimated Effect (+/-)[5]	References[6]
		1° Inoculum/ Disease Estab.	Rate of Disease Progress	Time[3]			
FIELD CROPS							
Alfalfa							
Anthracnose	*Colletotrichum trifolii* (F)	↑	↓	-	B / E O	-	a
Damping off	*Pythium* spp.(F)	-	↓	-	B / E K	--	a
Root rot	*Phytophthora megasperma* (F)	-	↑	-	B / E K	--	a
Verticillium wilt	*Verticillium albo-atrum* (F)	↑	↓	-	B/ G H.	+	a
Corn							
Anthracnose	*Colletotrichum graminicola*(F)	↑	↓	↑	B / E	-	b
Common smut	*Ustilago maydis* (F)	↑	↑	↑	F L	++	b
Eyespot	*Kabatiella zeae* (F)	↑	↓	↑	E	--	b
Fusarium ear rot	*Fusarium* (F)	↑	↑	↑	B / F L	+	b
Grey leaf spot	*Cercospora zeae-maydis* (F)	↑	↑	↑	B / E M	+	b
N. leaf blight	*Exserohilum turcicum* (F)	↑	↓	↑	B / E	-	b
* Stewart's wilt	*Erwinia stewartii* (B)	↑	↑	↑	C / G H	+	b
Soybean							
Brown stem rot	*Phialophora gregata* (F)	↑	↓	↑	A B / E G	-	c
Downy mildew	*Peronospora manchurica* (F)	-	↓	↑	D / E	-	c
Pod/stem blight	*Diaporthe phaseolorum* (F)	↑	↓	↑	B / M	-	c
Powdery mildew	*Microspheara diffusa* (F)	↑	↓	↑	B E	-	c
Root rot	*Phytophthora megasperma* (F)	↑	↓	↑	A / E G K	--	c
Root rot	*Rhizoctonia solani* (F)		↑	↑	A / G K	+	c
Stem canker	*Diaporthe citri* (F)	↑	↓	↑	B / E M	-	c
Sudden death	*Fusarium solani* (F)	-	↓	↑	A / E I	-	c
* White mould	*Sclerotinia sclerotiorum* (F)	↓	↓	↑	A / E K M	-	c
* Cyst nematode	*Heterodera glycines* (N)	↓	↑	↑	A / F G	++	c
Tobacco							
Blue mold	*Peronospora tabacina* (F)	-	↓	-	D / E J	-	d
Soybean mosaic virus (V)		-	↑	-	D / H I	+	d
Tobacco ringspot virus (V)		-	↑	-	D / H I	+	d
Winter Wheat							
Dwarf bunt	*Tilletia contraversa* (F)	-	↑	-	A	+	e
Head blight	*Fusarium* Sp. (F)	↑	↓	-	B / E M	-	e
Leaf spot	*Septoria* spp. (F)	↑	↓	-	B / K E M	--	e

Crop/Disease	Pathogen (Group)[1]	Anticipated Effect of Climate Change (↑,-,↓)[2]			Remarks[4]	Estimated Effect (+/-)[5]	References[6]
		1° Inoculum/ Disease Estab.	Rate of Disease Progress	Time[3]			
Winter Wheat (con't)							
Leaf rust	*Puccinia recondita* (F)	↑	↓	-	B D / E M	--	c
Loose smut	*Ustilago tritici* (F)	-	↓	-	D / E	-	c
Seedling blight	*Fusarium* spp. (F)	-	↑	-	D / G	+	c
Snow mould	*Microdochium nivale* (F) *Typhula* spp. (F) *Sclerotinia borealis* (F)	↓	↓	-	B	--	c
Stem rust	*Puccinia graminis* (F)	-	↓	-	D / E M	-	c
Take-all	*Gauemannomyces graminis*(F)	↑	-	-	B / E G K	-	c
Tan spot	*Pyrenophora tritici-repentis* (F)	↑	↓	-	B / E M	-	c
VEGETABLE CROPS							
Bean							
*Angular leaf spot	*Phaeoisariopsis griseola* (F)	↑	↓	-	B D / E	-	f
Damping-off	*Pythium* spp. (F)	-	↓	-	A / K	-	f
Gray mould	*Botrytis cinerea* (F)	-	↓	-	A B / M	-	f
Pod/leaf spot	*Alternaria alternata* (F)	↑	↓	-	B / E	-	f
White mould	*Sclerotinia sclerotiorum* (F)	↑	↓	-	A / E K	--	f
Bacterial blight	*Xanthomonas campestris* (B)	-	-	-	D / E F M	o	f
Bean common mosaic virus (V)		-	↑	-	D / H I	+	f
Bean yellow mosaic virus (V)		↑	↑	-	C / H I O	++	f
Carrot							
Cavity spot	*Pythium* spp. (F)	-	↓	-	A / E K	-	g
Crown rot	*Rhizoctonia solani* (F)	-	↓	-	A / E K	-	g
Leaf blight	*Alternaria dauci* (F)	↑	↓	-	B / E M	--	g
* Sclerotinia mould	*Sclerotinia sclerotiorum* (F)	↓	↓	-	A / E K	-	g
* Aster yellows	Phytoplasma (P)	↑	↑	-	B C / G I J	+	g
Onion							
Basal rot	*Fusarium oxysporum* (F)	↑	↑	-	A / F	+	g h
Damping-off	*Pythium* spp. (F)	-	↓	-	A / E K	--	g h
	Fusarium spp. (F)	↑	↑	-	A B C / G	+	
Downy mildew	*Peronospora destructor* (F)	-	↓	-	D / E M	-	g h
Leaf blight	*Botrytis squamosa* (F)	-	↓	-	A B D / E M	-	g h
Pink root	*Phoma terrestris* (F)	↑	↑	-	A / F G H	+	g h

Crop/Disease	Pathogen (Group)[1]	Anticipated Effect of Climate Change (↑,-,↓)[2]			Remarks[4]	Estimated Effect (+/-)[5]	References[6]
		1° Inoculum/ Disease Estab.	Rate of Disease Progress	Time[3]			
Onion (con't)							
Smut	*Urocystis colchici* (F)	-	↓	-	A / E	-	g h
White rot	*Sclerotium cepivorum* (F)	-	↓	-	A / E K	-	g h
Slippery skin	*Pseudomonas gladioli* (B)	-	↓	-	A / E L M	-	g h
Soft rot	*Erwinia caratovora* (B)	-	↑	-	A B / L M	+	g h
Sour skin	*Pseudomonas cepacia* (B)	-	↓	-	A / E L M	-	g h
Potato							
Canker	*Rhizoctonia solani* (F)	↑	↓	↑	A B / E	-	g i
Early blight	*Alternaria solani* (F)	↑	↓	↑	A B / E L	-	g i
Late blight	*Phytophthora infestans* (F)	↑	↓	↑	B D	-	g i
Pink rot	*Phytophthora erythroseptica* (F)	-		↑	B / K	-	g i
Verticillium wilt	*Verticillium* spp. (F)	↑	↑	↑	A / G	+	g i
Blackleg	*Erwinia carotovora* (B)	-	↓	↑	A B D / E K	-	g i
Common scab	*Streptomyces scabies* (B)	-	↑	↑	B D / K	+	g i
Ring rot	*Clavibacter michiganensis* (B)	↑	↓	↑	B D	-	g i
* Potato leaf-roll virus (V)		-	↑	↑	B C / H I	++	g i
Sweet Corn							
Common smut	*Ustilago zeae* (F)	↑	↑	↑	A B D / F L	++	b g
Ear rot	*Fusarium graminearum* (F)	↑	↑	↑	B / F L	+	b g
Leaf blight	*Exserohilum turcicum* (F)	↑	↓	↑	B / E	--	b g
Rust	*Puccinia sorghi* (F)	-	↓	↑	D / E J M	-	b g
Stalk rot	*Diplodia maydis* (F)	↑	↑	↑	B D / L	+	b g
	Fusarium spp. (F)	↑	↑		B / L	+	
	Pythium spp. (F)	-	↓		B / K	-	
* Stewart's wilt	*Erwinia stewartii* (B)	↑	↑	↑	C D / G H	++	b g
Tomato							
Anthracnose	*Colletotrichum coccodes* (F)	↑	↓	-	A B D/ L M	-	g j
Early blight	*Alternaria solani* (F)	↑	↓	-	B / E L	-	g j
Late blight	*Phytophthora infestans* (F)	↑	↓	-	B / E M	-	g j
Leaf spot	*Septoria lycopersici* (F)		↓	-	B D / E M	-	g j
Verticillium wilt	*Verticillium* spp. (F)	-	↑	-	B D / G	+	g j
Bacterial canker	*Clavibacter michiganensis* (B)	-	↑	-	B D / F L M	+	g j
* Bacterial spot	*Xanthomonas axonopodis* (B)	↑	↓	-	B D / L M	o	g j

Crop/Disease	Pathogen (Group)[1]	Anticipated Effect of Climate Change (↑,-,↓)[2]			Remarks[4]	Estimated Effect (+/-)[5]	References[6]
		1° Inoculum/ Disease Estab.	Rate of Disease Progress	Time[3]			
Tomato (con't)							
* Bacterial speck	*Pseudomonas syringae* (B)	↑	↓	-	B D / L M	o	g j
Cucumber mosaic virus (V)		↑	↑	-	C / G H I	++	g j
Tomato mosaic virus (V)		-	-	-	H	o	g j
HORTICULTURAL CROPS							
Apple							
Apple scab	*Venturia inaequalis* (F)	↓	↓	-	B / E M	-	k
Black rot	*Botryoshpaeria obtuse* (F)	↑	↓	-	B / E M	-	k
Bitter rot	*Glomerella cingulata* (F)	↑	↓	-	B / E M N	-	k
Cedar-apple rust	*Gymnosporangium juniperi-virginianae* (F)	-	↓	-	D / E M	-	k
Collar rot	*Phytophthora cactorum* (F)	↑	↓	-	A B / K N	-	k
Fly speck	*Zygophiala jamaicensis* (F)	↑	↓	-	B	-	k
Powdery mildew	*Podosphaera leucotricha* (F)	↑	↑	-	B / F L	+	k
Fire blight	*Erwinia amylovora* (B)	↑	↓	-	B / E L M N	o	k
Grape							
Black rot	*Guignardia bidwellii* (F)	↑	↓	-	B / M	-	l
Bunch rot	*Botrytis cinerea* (F)	↑	↓	-	B / E L	-	l
Cane rot	*Phomopsis viticola* (F)	↑	↓	-	B / M N	-	l
Dead arm	*Eutypa lata* (F)	↑	↓	-	B / E N	-	l
Downy mildew	*Plasmopara viticola* (F)	↑	↓	-	B	-	l
Powdery mildew	*Uncinula necator* (F)	↑	↑	-	F	+	l
Crown gall	*Agrobacterium tumefaciens* (B)	-	-	-	A C / N	o	l
Peaches							
Brown rot	*Monilinia fructicola* (F)	↑	↓	-	B / E L N	-	m
Peach canker	*Cytospora* spp. (F)	↑	↓	-	B / E G L N	+	m
Plum pox virus (V)		-	↑	-	C / H I	+	m
Strawberries							
Anthracnose	*Colletotrichum* spp. (F)	↑	↓	-	B C / E	-	n
Common leaf spot	*Mycosphaerella fragariae* (F)	↑	↓	-	A B / E M	-	n
Gray mould	*Botrytis cinerea* (F)	↑	↓	-	A B / E	-	n
Leaf blight	*Phomopsis obscurans* (F)	↑	↓	-	B/ M O	-	n
Leaf scorch	*Diplocarpon earliana* (F)	↑	-	-	B / O	o	n
Leather rot	*Phytophthora cactorum* (F)	-	↓	-	A / E K O	--	n

Crop/Disease	Pathogen (Group)[1]	Anticipated Effect of Climate Change (↑,-,↓)[2]			Remarks[4]	Estimated Effect (+/-)[5]	References[6]
		1° Inoculum/ Disease Estab.	Rate of Disease Progress	Time[3]			
Strawberries (con't)							
Powdery mildew	*Sphaerotheca macularis* (F)	↑	↑	-	B / F	+	n
Angular leaf spot	*Xanthomonas fragariae* (B)	↑	↓	-	B / E M	--	n
Root lesion	*Pratylenchus* spp. (N)	-	↑	-	A / G	+	n
Turf							
Anthracnose	*Colletotrichum graminicola* (F)	↑	↑	-	B / G	+	o
Brown patch	*Rhizoctonia solani* (F)	-	↑	-	B / G F	-	o
Dollar spot	*Sclerotinia homoeocarpa* (F)	↑	↑	-	B / G F	+	o
Fusarium patch	*Microdochium nivale* (F)	↑	↓	-	B / E	-	o
Grey snow mould	*Typhula* spp. (F)	↑	↓	-	AB / E	--	o
Melting out	*Drechslera* spp. (F)	↑	-		B	-	o
Pink snow mould	*Microdochium nivale* (F)	↑	↓	-	B / E	-	o
Powdery mildew	*Erysiphe graminis* (F)	↑	-	-	B	o	o
Cottony blight	*Pythium aphanidermatum* (F)	↓	↓	-	A / E G	-	o
Root rot	*Pythium* spp. (F)	↓	↓	-	A / E G	-	o
Necrotic ring spot	*Leptosphaeria korrae* (F)	↑	↓	-	B / E G	-	o
Red thread	*Laetisaria fuciformis* (F)	-	↓	-	A / E	-	o
Rust	*Puccinia* spp. (F)	↑	↑	-	B D	+	o
Summer patch	*Magnaporthe poae* (F)	↑	↓	-	B	-	o
Take-all patch	*Gaeumannomyces* (F)	↑	↓	-	B / G K	+	o
FOREST TREES							
Pine							
Armillaria root disease[7]	*Armillaria* spp. (F)	-	↑	↑	F	+	p r t
Blister rust	*Cronartium ribicola* (F)	↓	↓	↓	E M	-	q r
Blue stains	*Ophiostoma* spp. (F)	↑	↑	-	C / F H	+	s t
Diplodia tip blight	*Sphaeropsis sapinea* (F)	-	↑	-	G	+	r t
Fomes root rot	*Heterobasidium annosum* (F)	↑	-	↑	B / F N	++	r t
Scleroderris canker European race	*Gremmeniella abietina* (F)	↓	↓	-	E	-	r u
Pine wood nematode	*Bursaphelenchus xylophilus* (N)	-		↑	F G H	+	t
Dwarf mistletoe[8]	*Arceothobium americanum* (PL)	-	-	↑	F	+	v

Crop/Disease	Pathogen (Group)[1]	Anticipated Effect of Climate Change (↑,-,↓)[2]			Remarks[4]	Estimated Effect (+/-)[5]	References[6]
		1° Inoculum/ Disease Estab.	Rate of Disease Progress	Time[3]			
FOREST TREES (con't)							
Spruce							
Tomentosus root rot	*Inonotus tomentosus* (F)	-	↑	-	F	+	r t
Larch							
Larch canker[8]	*Lachnellula willkommii* (F)	↓	↓	-	E	-	r t
Poplar							
Conifer-aspen rust	*Melampsora medusae* (F)	↓	↓	-	M	-	w
Hypoxylon canker	*Entoleuca mammata* (F)	↑	↑	-	L	+	w
Oak							
Oak wilt[8]	*Ceratocystis fagacearum* (F)	↑	↑	↑	F I K	+	
Beech							
Beech bark	*Nectria coccinea faginata* (F)	↑	↑	-	H N	+	x
Elm							
Dutch elm disease	*Ophiostoma ulmi* (F)	↑	↑	-	C F I	+	y
Forest declines							
ash		↑	↑	-	G K	++	z
* maple		↑	↑	-	G K	++	z
oak		↑	↑	-	G K	++	z

[1] Pathogen group: B = bacteria; F = fungus; N = nematode; P = phytoplasma; V = virus; PL = parasitic plant.

[2] ↑= Anticipated increase, ↓= decrease, and - = no change.

[3] Anticipated effect of increased growing season length on disease.

[4] Reason(s) for anticipated effect:

A. Initial or primary inoculum is soilborne and expected to remain at the same levels, or possibly decrease, due to milder winters, longer growing season, and increased microbial competition.

B. Initial or primary inoculum is debrisborne or survives on host and is expected to increase due to increased survival over milder winters.

C. Initial or primary inoculum is insectborne and is expected to increase due to increased survival of insect vectors.

D. Initial or primary inoculum is introduced each year from outside sources i.e., seedborne, airborne. The influence of climate change is difficult to assess without knowledge of the pathogen, host, and source of inoculum.

E. Warmer and/or drier growing season slows rate of disease progress.

F. Warmer and/or drier growing season increases rate of disease progress.

G. Increased severity of disease symptoms due to stress of drier and/or warmer summer growing conditions.

H. Increased survival of insect vectors due to milder winter.

I. Increased rate of development of insect vectors due to warmer temperatures.

J. Earlier introduction of vectors or pathogens from southern regions.

K. Reduced soil moisture due to increased evapotranspiration, sporadic precipitation, etc., affects pathogen.

L. Increased wound sites on hosts due to increased extreme weather events (i.e., thunderstorms, high winds, hail) and /or increased insect damage increases infection sites.

M. Reduced disease development and spread due to decrease in rain and/or length of time of leaf wetness.

N. Fruiting bodies on trees have a longer active growth period in both fall and spring, hence more primary inoculum.

O. On non-woody perennials, pathogens have longer to grow on roots or overwintering leaves so more damage and perhaps increased primary inoculum in spring.

[5] Net anticipated effect on a particular plant disease. From a significant increase (++) to a significant decrease (--) in importance (--, -, o, +, ++).

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~ Affects all tree species.

§ In North America but not yet in Ontario.

* There is a case study of this disease/pathogen in the text of the report.

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